THE
LARYNGOSCOPE.

VOL. LXIX OCTOBER, 1959 No. 10

A STUDY OF THE FATE OF AUTOGENOUS CARTILAGE GRAFTS.*

MORRIS DAVIDSON, M.D.,

St. Louis, Mo.

I. INTRODUCTION.

The present study was done to investigate generally, the fate of autogenous cartilaginous transplants in the external ears of animals, and to determine specifically whether the union that takes place between a carefully approximated autogenous graft and its host cartilage is fibrous or whether new cartilage is formed. It was felt that this study may give some clue as to the fate of the cartilaginous portion of a composite graft from the ear of humans for transplantation to defects in the external ear, ala nasae, and columella. A composite graft is defined as one that contains more than one type of tissue, such as both cartilage and skin. In this instance, however, only the cartilaginous portion is being considered. The cartilage of the ear of the rabbit was selected, because it so closely resembles the elastic cartilage in the ears of humans.

According to the microscopic appearance and nature of its fibrillar components, three varieties of cartilage are distinguished: hyaline, elastic, and fibrous. Hyaline cartilage has the widest distribution throughout the body and is characterized by a matrix that is almost clear and translucent and is free from obvious connective tissue fibers. Elastic cartilage

*Submitted as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1958.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 28, 1959.

possesses cells like those of hyaline cartilage, but the matrix contains numerous branching elastic fibers. Elastic cartilage is found in the external ear, ear canal, Eustachian tube, and epiglottis. Fibrocartilage has a cellular structure like hyaline and elastic cartilage, but the matrix contains thick, compact collagenous bundles which lie parallel to one another. Fibrocartilage is found in the intervertebral discs, symphysis pubis, and the interarticular menisci of many joints.

The voluminous literature dealing with cartilage grafts is, for the most part, contradictory and confusing as regards any conclusions. This is primarily due to the variations in the different experimental procedures utilized. The autogenous grafts of cartilage have, for instance, been subjected by many investigators to removal of the perichondrium, to boiling, freezing, dehydration, etc. The laboratory animals commonly used have been rabbit, chicken, mouse, guinea pig, and dog. Grafts have been taken from rib, ear, joint, sternum, and other sites and inserted in subcutaneous tissues, intramuscular areas, anterior chamber of the eye, and joint synovial cavities.

A summary in chronological order of the more pertinent findings reported in the literature is presented.

II. REVIEW OF THE LITERATURE.

1. Literature Previous to the Twentieth Century.

The earliest writers were primarily concerned with the healing of wounds or injuries to cartilage. Hunter[1] (1743), Dorner[2] (1798), Meckel[3] and Pauli[4] (1854), and Leidy[5] (1849) agreed that cartilage had no tendency to regenerate or unite after injury. Redfern,[6] who in 1851 used a microscope for observations, reported that healing of wounds in rib and ear cartilages was accomplished by ingrowth of fibrous tissue and some activity on the part of the cartilage cells at the edges of the wound. Two years later, in 1853, Kolliker[7] and Paget[8] found no evidence that fragments of cartilage united after injury or rupture, and further stated that new cartilage does not replace damaged cartilage. Bert[9] (1865) was presumably the first to report viability of cartilage with trans-

plantation. He buried a rat's tail beneath the skin as an autogenous graft and noted that the cartilage remained viable for months. Ollier[10] (1867) was in agreement with Bert and further stated that adult animal cartilage will survive when transplanted if the perichondrium remains attached to the graft. Fisher[11] (1882) agreed that perichondrium was necessary for survival of a graft. Zahn[12] (1884) believed that all cartilage grafts tended to undergo degeneration regardless of whether they were homogenous or autogenous. On the other hand, Prudden[13] in 1887, substantiated Bert's observations, and since he removed the perichondrium from the transplants apparently proved that cartilage can survive without the perichondrium. He transplanted small pieces of hyaline cartilage to the subcutaneous tissue of rabbits and allowed them to remain in place as long as 399 days. He believed the cells lived for many months and even changed in size and shape.

The first use of cartilage transplants in humans is credited to Konig[14] (1896), who used them to repair tracheal defects with successful results.

At the beginning of this Century the interest turned more intently to the histological changes responsible for healing of wounds in cartilage..

2. Literature from 1900 to the Present.

Matsuoka[15] (1904) and Mori[16] (1905) agreed that cartilage injuries healed by the proliferation of a layer of connective tissue from the perichondrium which became partly changed into fibrocartilage. Seggel,[17] (1904) however, was of the opinion that cartilage cells multiply by mitosis. Haas[18] (1914) concurred with Matsuoka and Mori in stating that regeneration of cartilage was from the perichondrium. Fisher[19] (1922) seemed to agree with Seggel, as he found that superficial cartilage cells became the parent cells of the more fully developed cartilage in its deeper parts. Ito[20] (1924) further substantiated the work of Matsuoka and Mori. He worked with the articular cartilage of rats and found that defects in the grafts were first filled with granulation which in time changed into fibrous tissue, then to fibrocartilage, and finally into cartilage. It would appear that the writers in the early

part of the Twentieth Century were in agreement that cartilage does tend to regenerate following injury. There was no agreement concerning the process by which healing took place.

The work of Loeb[21] (1926) remains a classic to this day. He found that cartilage and its attached structures such as perichondrium, fat, and areolar tissue remain well preserved after autotransplanation. Homotransplants of cartilage, on the whole, remained well preserved. It was his contention that the perichondrium may form new cartilage in both instances. He noted a great difference in cellular response evoked by autotransplants of cartilage as compared with homotransplants of cartilage. After autotransplantation, lymphocytes were almost entirely lacking, whereas in the homotransplant there was a considerable lymphocytic response. He found that lymphocytes entered necrotic areas of cartilage and replaced them, but that they migrated only to a very limited degree into living, well-differentiated cartilage where they soon perished. Lymphocytic invasion became enhanced if the intercellular substance was decreased.

Key[22] (1931) was of the opinion that the repair of defects in hyaline cartilage occurred through the proliferation of fibrous tissue with transformation into cartilage. Publishing in the same year, Shands[23] was largely in agreement with Key. He stated that following injury, fibrin was first laid down, to be followed in turn by granulation tissue and connective tissue. Cartilage cells that began to form in the connective tissue became fibrocartilage which developed into new hyaline cartilage. A year later, Fell[24] stated that perichondrial cells possessed the specific power to form cartilage cells.

It was in 1939 that Peer[25] published the first of his excellent and voluminous works with cartilage grafts. After burying autogenous rib cartilage grafts for four-and-a-half and six years he found them to be living cartilage when removed. He concluded that this type of graft survived up to six years and remained the same size. Dupertius[26] (1941) demonstrated the actual growth of transplanted young cartilage and believed that the increase in size proved the viability of the transplant. His studies were further extended, and in 1950[38] he demonstrated a measurable growth of autogenous cartilage

transplants in humans. This was accomplished by inserting a piece of rib cartilage into the dorsum of the nose of young children. He reported an increase in length of the graft that was measurable. In addition, he was of the opinion that perichondrium was not necessary for the growth.

Clark and Clark[27] by means of their transparent chamber installed in the rabbit's ear, observed the late formation of new cartilage. After vascularization of the area was completed new cartilage cells were seen to arise from elongated motile cells. The latter contained uniformly distributed granules that enlarged and coalesced to form fat droplets similar to those seen in the intact ear cartilage and matrix which appeared simultaneously between the cells. They were unable to determine the origin of the new cells but believed they were either from the original cartilage and perichondrium or from fibroblasts arising from the connective tissue. The fact that the new cartilage was spotty in distribution was interpreted as probably indicating a stimulus of the nature of a localized chemical change in the tissues. In addition, there was occasionally some development of new bone in the midst of an area of new cartilage.

Young[28] (1941) found that autogenous rib cartilage grafts to the abdomen remain viable. In all of his specimens the cellular structure was found to be intact, the cell nuclei were present, and the grafts were pliable and retained their spring-like elasticity. In another experiment, Young placed fine particles of chopped cartilage into the abdominal wall without or with perichondrium and found that all the particles were viable on removal and had become adherent to one another by fibrous tissue.

Peer[29] in 1944 wrote that autogenous cartilage is the material of choice for transplantation in that it survives as living tissue; is not subject to invasion or absorption, and remains as part of the living organism. He emphasized that the matrix of cartilage gives this structure its property of elasticity which adapts it for special function or support. The ultimate survival of a graft is determined by the living cartilage cell since this cell produces the matrix. In addition to proving the viability of his grafts, Peer[30] writing two years later sub-

stantiated the work of Dupertius, but in the human. He took measured pieces of young autogenous grafts from the rib, nasal septum, and ear, with the perichondrium removed, and buried them for intervals up to two years. There was a definite increase in size of the transplanted tissues.

Brown, et al.,[31,32,33] presented three papers in 1946, dealing with the successful use of composite grafts of skin and cartilage from the ear in humans to replace the loss of tissues from such areas as the nostril border, nasal tip, and columella. Since the original paper by Brown in 1946 other papers presenting the successful use of composite grafts have been published: Brown[34] (1947), Wachsberger[35] (1947), Szlazak[36] (1949), Converse[37] (1950), Duformental[39] (1951), Farina[41] (1952), and McLaughlin[49] (1954). Brown states that the transplanted cartilage does persist and can be felt in the grafted area months after surgery.

Peer and Walker[40] (1951) found that free human cartilage autografts in contact with like tissues retain their specific structures and that cartilage autografts in contact with unlike tissues can survive transplantation unchanged and retain their same relative size. It was their finding that cartilage transplants in the human are joined to the host cartilage by fibrous union and not by cartilaginous union.

Laskin and Sarnat[43] wrote the first of their articles on the respiration and anaerobic glycolysis of transplanted cartilages in 1952. They found the rate of respiration and anaerobic glycolysis of fresh rabbit costal cartilage to be among the lowest of all tissues. They determined that cartilage metabolism is predominantly anaerobic, and that transplantation resulted in a decline of approximately 45 per cent in the rates of both respiration and anaerobic glycolysis in both autogenous and homogenous grafts during the first seven days. They could find no difference after seven to 150 days between metabolic activity of costal cartilage autogenous grafts or homogenous grafts. In papers published in 1953 and 1954, Laskin and Sarnat[44,45] found that cartilage grafts were able to survive because the metabolism of the grafts was very low, and thus the factor of complicated cellular and immunologic response of the host as occurs in grafts of higher metabolic response

was either eliminated or decreased. Because cartilage exhibits a predominantly anaerobic metabolism it can better withstand the hypoxic states during transplantation, and being avascular, it can more readily establish its normal diffusion pattern with the surrounding tissue fluids.

In the 1952 edition of their textbook, Maximow and Bloom[42] stated that after a wound or excision of a portion of living hyaline cartilage in adult mammals regeneration does not take place.

Longmire, et al.,[46] writing in 1954, believed there was almost universal agreement concerning the superiority of autogenous grafts over homologous tissue grafts. They felt that it was the general impression that autogenous grafts of all kinds are less susceptible to infection, inflammatory reaction, absorption, or other physical alteration than comparable homologous grafts.

Billingham[47] (1954) stated that the actual cells in cartilage grafts, even in freshly removed and transplanted grafts, die very soon after transplantation. The reason was presumably due to a delay in the penetration of host vessels into the grafts and, therefore, play practically no part in the healing process.

In 1954, Peer[48] wrote that normal growth of all cartilage structures during childhood takes place from the deep layers of connective tissue cells of the perichondrium which form a matrix about themselves, separate from the perichondrium and become cartilage cells. He stated that cartilage ceases to grow after adulthood is reached.

Rao[50] (1954) reported his experimental results on the regeneration of cartilage in rats. He incised the sternum in the midline and kept the divided parts in apposition by sutures. The healing process was studied at intervals. After two days the areolar tissue surrounding the cartilage was edematous. After four days the gap between the cartilage was filled with fibrin clot, cellular infiltrate with edema, and congestion of the fatty tissues. After seven days the fibrin clot underwent organization, edema was reduced and fibroblasts developed from fibrous fascia and perichondrium. After 15 days the fibrin was invaded by granulation tissue, and the cut ends

of the cartilage were irregular due to the outflow of hyaline matrix. After 28 days there was a cluster of new cells. Between 30 and 50 days the cut edge of the cartilage increased in size due to a greater amount of hyaline matrix and proliferation of cartilage cells. After 90 days the gap between the cut ends was filled with fibrocartilage and closely resembled young cartilage cells. Toluidine blue stain showed the matrix in stages of transformation into hyaline ground substance. At 114 days the new cells and matrix resembled original cartilage and differed only in the absence of cell arrangement of lacunar spaces and columns. The matrix lacked the brilliant color reaction with McManus's stain which suggested a deficiency of chondroitin sulphate. He believed the proliferation of cartilage cells occurred by mitosis. This process was slow, sporadic, and incomplete. He stated that the fibrous tissue was derived from the perichondrium as well as the surrounding connective tissue.

Gibson[51] (1955) was of the opinion that cartilaginus repair occurred in three possible ways: proliferation of chondrocytes, proliferation of vascular connective tissue from the perichondrial margins and by the ingrowth of vascular connective tissue.

Craigmyle[52] (1955) reported on experimental studies with cartilage grafts in rabbits and concluded that cartilage retained its structure after autotransplantation, and that grafts appeared to remain viable.

Peer,[53] in his excellent book of 1955, stated that there is no evidence that cartilage unites by other than fibrous union in humans.

III. METHOD APPROACH TO THE PROBLEM.

Five healthy adult white rabbits weighing approximately five pounds each were selected for this study. Anesthesia was obtained by the subcutaneous injection of pentobarbital sodium. In all, ten separate cartilage grafts were obtained for study for periods varying up to 150 days.

Each transplantation was prepared using aseptic surgical technique. The skin over the ear was shaved, cleansed with

soap and water and alcohol applied. Each area was draped with sterile towels. Fine forceps, small curved scissors, and small eye needles were used. Hemostasis was obtained by the use of small hemostats and fine silk sutures. The edges of the wound were carefully approximated with silk sutures.

An elliptical incision of about 15 mm. in the greatest diameter was made in the skin in the mid-portion of the ventral surface of the ear. The skin was carefully elevated with sharp dissection by scalpel and scissors, care being taken to avoid any injury to the underlying perichondrium. A previously sterilized cork-borer with a diameter of 7 mm. was employed to cut through the perichondrium on the ventral surface, the cartilage, and the perichondrium on the dorsal surface of the ear. Care was taken to avoid cutting the skin on the dorsal surface of the ear. The cork-borer was used because it could be readily sterilized and was easily sharpened. It cut the graft and the recipient bed of exactly the same diameter. The circular piece of cartilage was then removed from its bed by sharp dissection and placed in a similarly prepared bed in the opposite ear. The graft fitted snugly into the recipient area, and it was not necessary to employ sutures to hold it in place. All the animals tolerated the procedure well.

The animals were sacrificed at intervals of 30 days. An incision through the skin was made and another circular piece of cartilage and its attached perichonrium was removed. At this time, however, the piece of cartilage removed was approximately 3 mm. wider at its periphery than the original graft to include the point of union and the surrounding cartilage.

The specimens were fixed in 10 per cent formalin for four to seven days. They were then rinsed in water and dehydrated in alcohols (50 per cent, 70 per cent, 80 per cent, 90 per cent, 95 per cent, and absolute), for three to four hours in each, following which each was cleared in cedarwood oil and finally infiltrated and embedded in 56° C. paraffin. Sections of 5 to 10 microns in thickness were made through the specimen in a horizontal plane. They were mounted and then stained with Harris hematoxylin and eosin.

IV. FINDINGS.

Macroscopic examination of the grafts on their removal prior to fixation, staining, and sectioning revealed them to be quite flexible and elastic. There was no visible difference in texture between the graft and surrounding cartilage. In the 30, 60, and 90 day grafts there was evidence of a slight cir-

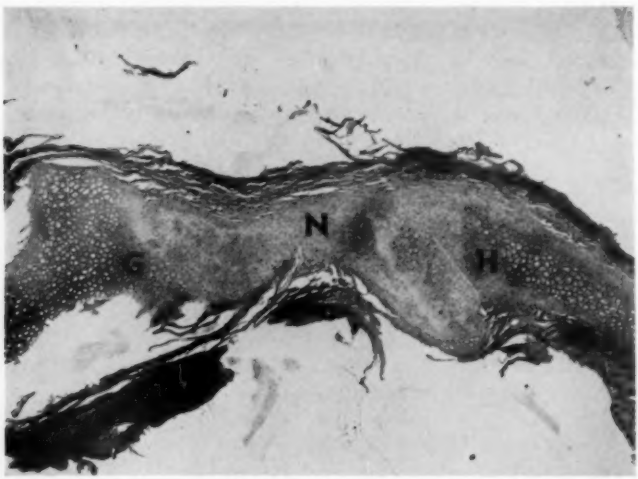

Fig. 1. A photomicrograph of a section through the junction between the host and graft cartilages after 30 days. The union at this time, for this specimen, is cartilaginous. To the left is the graft (G). The cartilage is in a viable state although it is not particularly adherent to the surrounding connective tissue. To the right is the host cartilage (H) with perichondrium and surrounding tissues intact. There is proliferation of perichondrial cells on either side of the host cartilage for a short distance and it is presumed that most of the union between transplant and host is from the intact perichondrium of the host. Note the irregular arrangement and size of new cartilage cells (N) and the tendency for the new cells to occur in groups with varying amounts of matrix about them. Six microns in thickness; Harris's hematoxylin and eosin; 25X.

cular cut. In the 120 and 150 day grafts, the graft and the host cartilage appeared to be continuous.

The junction between host and transplant cartilages after 30 days is illustrated in Fig. 1. Young cartilage cells unite the two tissues. There is evidence that the perichondrium of the host cartilage has been the chief factor in the production

of the newly formed cartilage. The thickened and active perichondrium of the host cartilage is in contrast to that of the graft. At this stage the subcutaneous tissues on either side of the graft cartilage have not become firmly attached to it.

Fig. 2 shows an area of new bone formation occurring in

Fig. 2. A photomicrograph of the union between host and transplant cartilages after 60 days. The host cartilage (H) is to the left and the graft (G) is to the right. Between the two is a cartilaginous and fibrous union in which new bone formation (B) has occurred. The perichondrium on the side of the host cartilage shows considerable activity and the formation of new cartilage cells. The perichondrium of the graft cartilage, while relatively inactive, is closer to the new bone which encircles a more vascular area. The graft at this stage is firmly attached to the surrounding connective tissue. Six microns in thickness; Harris's hematoxylin and eosin; 25X.

the new cartilage after 60 days. Fibrous tissue is seen to unite the host and graft cartilages. There are a number of small blood vessels in the area encircled by bone. It appears to have a rather dense fibrous connective tissue about it. The proliferation of new cartilage cells from the perichondrium of the host cartilage is in contrast to the less active perichondrium of the graft. The latter cartilage has maintained its

viability, and the surrounding connective tissue is firmly attached to it.

A somewhat higher power view of the union between the graft and host cartilages after 90 days is shown in Fig. 3. Most of the newly formed cartilage cells are small with more matrix between the cells than is seen in the 30-day and 60-day

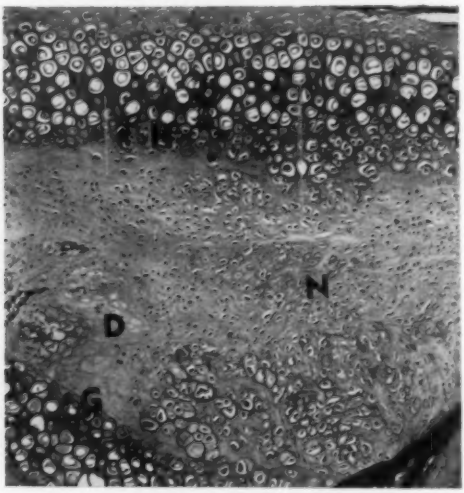

Fig. 3. A photomicrograph of the union between graft and host cartilages after 90 days. The rapidly dividing cartilage cells (N) are for the most part from the perichondrium of the recipient cartilage (H) while degenerated cartilage cells (D) can be seen on the border of the transplanted cartilage (G). Many of the new cartilage cells are bi- or multinucleated and irregularly arranged. Six microns in thickness; Harris's hematoxylin and eosin; 60X.

specimens. On the border of both host and graft cartilages an area of irregularly arranged and larger cells appear to be undergoing rapid division. There is, in addition, an area designated by "D" which shows degenerative changes in presumably mature cells of the graft cartilage. This type of alteration was found to occur in most of the specimens but never involved the greater part of the graft, and was usually a small area located on the periphery.

The junction between host and graft cartilages after 120 days is shown in Fig. 4. There is nothing significantly different from this specimen and previously shown specimens in other figures except the formation of new bone which seems to be encroaching on an area of newly formed cartilage. There are a number of small blood vessels in relation to the new bone, but in contrast to Fig. 2 the new bone is not completely surrounded by dense fibrous connective tissue.

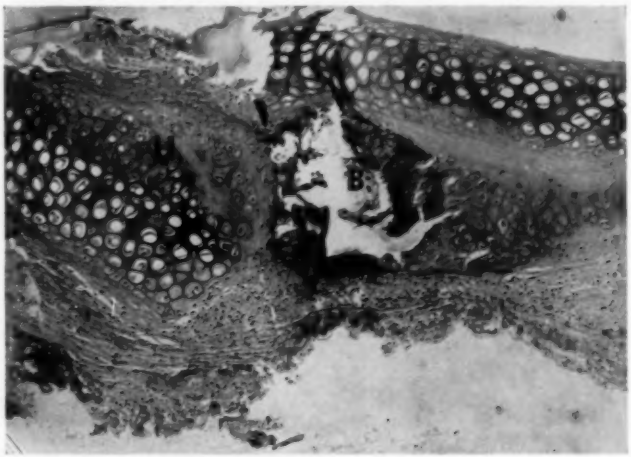

Fig. 4. A photomicrograph of the union between graft (G) and host cartilages (H) after 120 days. The cartilaginous union is being encroached upon by some new bone formation (B) which appears to be in a rather highly vascularized region. Six microns in thickness; Harris's hematoxylin and eosin; 65X.

A firm cartilaginous union between host and graft cartilages is apparent after 150 days and shown in Fig. 5. The union is comprised of cells which are more mature than those in the preceding figures, but the matrix stains less intensely than that of the host and graft cartilages.

V. DISCUSSION.

The results presented show that in the external ear of the experimental animals cartilaginous union occurs between the

graft and host cartilage when the borders of each are closely approximated.

It is well established that autogenous cartilage grafts retain their viability. The early writers, Bert and Prudden, and the later studies of Loeb, Dupertius, Peer, and others have proven conclusively that autogenous cartilage does live after

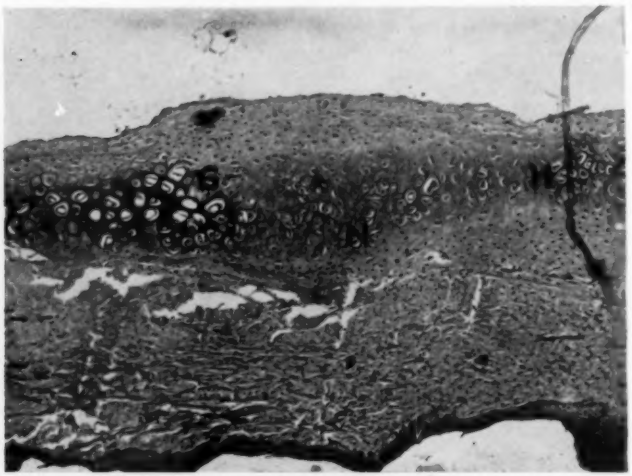

Fig. 5. A photomicrograph of the cartilaginous union between the graft (G) and host cartilages (H) after 150 days. The new cartilage cells (N) have some semblance of arrangement with more matrix interening between the cells although the cartilage is still immature. Six microns in thickness; Harris's hematoxylin and eosin; 65X.

transplantation. In the present study the viability of the grafts was definitely established. Macroscopically the grafts retained their normal appearance and elasticity. Macroscopically the cartilage cells retained their nuclei, matrix, and typical structure.

The work of Young, Dupertius, and Peer has proven that the presence of attached perichondrium is not necessary for the survival of the autogenous cartilage graft. In the present work, the attached perichondrium was required to complete the experiment, and hence no comparison can be made.

In this study, no measurements, were taken following the removal of the graft and, therefore, no data is available concerning the quantitive growth of the cartilage. It has been pointed out, however, that there was no shrinkage of the graft, and in the 120-day and 150-day specimens it was impossible to determine the exact site of contact between the graft and the host area by macroscopic means. Dupertius and Peer have already demonstrated that young cartilage cells have the ability to grow following transplantation.

The work of Laskin and Sarnat, with the respiration and anaerobic glycolysis of transplanted cartilage sheds much light on the factors involved in the viability of cartilage grafts. They demonstrated that the low metabolic rate and the largely anaerobic metabolism of cartilage grafts permits the tolerance of the hypoxic states during the early period of transplantation.

The present study is in complete agreement with the findings of Loeb. Very little lymphocytic reaction was produced and there was no evidence of any foreign body reaction surrounding the cartilage grafts. This indicates again that autogenous cartilage grafts evoke very little cellular response and also attests to the lack of sepsis in the wound area.

It is interesting to speculate concerning the origin and formation of new bone which occurred in two of the specimens. Clark and Clark found, in the transparent chamber of the rabbit's ear a similar development of new bone in the midst of an area of new cartilage. It is possible that a slight change of unknown origin in the stimulus of the substance forming new cartilage may account for the formation of the new bone.

Young found that finely chopped particles of cartilage embedded in the abdominal wall became adherent by fibrous union. Brown has stated that autogenous cartilage grafts retain their integrity, but there was no statement concerning the specific type of union that occurs between the graft and its host area. Peer and Walker state that cartilage transplants in the human are joined to the host cartilage by fibrous union and not by cartilaginous union. There is no question that the specimens obtained in this study are joined firmly

and closely as determined both macroscopically and microscopically. It is probable that this cartilaginous union was obtained because of the care taken to insure exact approximation between the host and the graft. There is no doubt that the presence of an intact perichondrium and established blood supply of the host area have contributed to and hastened the union. A careful review of the literature reveals no experimentation which is identical to this particular study.

VI. CONCLUSIONS.

1. Cartilaginous union between the graft and host cartilage occurred.

2. Small areas of bone formation occurred to some extent in two of the specimens (60 days and 120 days) at the point of union.

3. The perichondrium of the host cartilage proliferates and produces new cartilage which is underway in 30 days.

4. The perichondrium of the graft is also active, but less so, at the point of union with the host cartilage.

5. The older cartilage cells at the border of the graft appear to undergo degenerative changes with encroachment of the newly formed cartilage.

6. The remainder of the cells of the graft retain their viability, and at 150 days there was no evidence of a replacement over a 90-day specimen.

7. It is possible that the cell degeneration at the periphery of the graft may be due to tissue injury while the cells more centrally located maintain their integrity.

8. Vascularity was found to be moderate in the areas of the ears from which the grafts were taken and also transplanted. In the early stages there was some proliferation of fibrous tissue and small blood vessels into the region of contact of the graft with the host area; following this, perichondrial cells were seen to proliferate and new cartilage formation occurred. In the rapidly forming areas of new cartilage, binucleated cells were markedly in evidence.

BIBLIOGRAPHY.

1. Hunter, W.: On the Structure and Diseases of Articulating Cartilages. *Philo. Trans. Roy. Soc. London*, 9:267, 1743.

2. Dorner, Cited by Marchand, F.: Der Process Der Wundheiling. *Deutsche Chirurgie*, 16:268, 1901.

3. Meckel, cited by Dupertius.[28]

4. Pauli, F., cited by Marchand[2] and Dupertius.[28]

5. Leidy, J.: On the Intimate Structure and History of Articulating Cartilage. *Amer. Jour. Med. Sci.*, 17:277, 1849.

6. Redfern, P.: On the Healing of Wounds in Articulating Cartilage. *Month. Jour. Med. Sci.*, Edinburgh, 13:201, 1851.

7. Kolliker, A.: "Manual of Human Histology," 1:59. Sydenham Soc., London, 1853.

8. Paget, J.: Healing of Cartilage. *Lectures on Surgical Pathology*, London, 1:263, 1853.

9. Bert, P.: Sur la Greffe Animale. *Compt. Rend. Acad d. Sci.*, 61:587, 1865.

10. Ollier, L.: "Traite Experimental et Clinique de la Regeneration des Os et de la Production Artificielle du Tissue Osseux," 1:162. V. Masson et fils, Paris, 1867.

11. Fischer, E.: Ueber Transplantationen von Organischem Material. *Deutsche Ztschr. f. Chir.*, 17:362, 1882.

12. Zahn, F.: Ueber das Schicksal der in den Organismus Implantierten Gewebe. *Virchow's Arch. f. Path. Anat.*, 95:369, 1884.

13. Prudden, T. M.: Experimental Studies on the Transplantation of Cartilage. *Amer. Jour. Med. Sci.*, 82:360-370, 1887.

14. Konig, F.: Zur Deckung von Defecten in der Vorderen Tracheealwand. *Berlin Klin. Wochenschr.*, 33:1129-1139, 1896.

15. Matsuoka, M.: The Regeneration of Cartilage. *Virchow's Arch. f. Path. Anat.*, 175:33-45, 1904.

16. Mori, M.: Studies on Cartilage Regeneration. *Deutsche Ztschr. f. Chir.*, 76:220, 1905.

17. Seggel, R.: Experimentelle Beitrage zur Anatomie und Pathologie des Gelenkknorpels. *Deutsche Ztschr. f. Chir.*, 75:453, 1904.

18. Haas, S. L.: Regeneration of Cartilage and Bone with a Special Study of These Procedures as They Occur at the Chondrocostal Junction. *Surg., Gynec. and Obst.*, 19:604-617, 1914.

19. Fisher, A. G.: A Contribution to the Pathology and Etiology of Osteoarthritis. *Brit. Jour. Surg.*, 10:52, 1922.

20. Ito, L. K.: Nutrition of Articular Cartilage and its Method of Repair. *Brit. Jour. Surg.*, 12:31, 1924.

21. Loeb, L.: Autotransplantation and Homoiotransplantation of Cartilage in the Guinea Pig. *Amer. Jour. Path.*, 2:111-122, 1926.

22. Key, J. A.: Experimental Arthritis: Changes in Joints Produced by Creating Defects in Articular Cartilages. *Jour. Bone and Joint Surg.*, 13:725-739, 1931.

23. Shands, A. R.: The Regeneration of Hyaline Cartilage in Joints. *Arch. Surg.*, 22:137-178, 1931.

24. Fell, H. B.: Chondrogenesis in Cultures of Endosteum. *Proc. Roy. Soc.*, 112:417-427, 1932.

25. Peer, L. A.: The Fate of Living and Dead Cartilage Transplanted in Humans. *Surg., Gynec. and Obst.*, 68:603-609, 1939.

26. Dupertius, S. M.: Actual Growth of Young Cartilage Transplants in Rabbits. *Arch. Surg.*, 43:32-63, 1941.

27. Clark, R. E., and Clark, E. L.: Microscopic Observations on New Formations of Cartilage and Bone in the Living Mammal. *Amer. Jour. Anat.*, 70:167-200, 1942.

28. Young, F.: Autogenous Cartilage Grafts. *Surg.*, 10:7-20, 1941.

29. Peer, L. A.: Cartilage Grafting. *Surg. Clin. N. Amer.*, 24:404-419, 1944.

30. Peer, L. A.: Experimental Observations of Growth of Young Human Grafts. *Plast. and Reconst. Surg.*, 1:108-112, 1946.

31. Brown, J. B., and Cannon, B.: Composite Free Grafts of Skin and Cartilage from the Ear. *Surg., Gynec. and Obst.*, 82:253-255, 1946.

32. Brown, J. B., and Cannon, B.: Composite Free Grafts of Two Surfaces of Skin and Cartilage from the Ear. *Ann. Surg.*, 124:1101-1107, 1946.

33. Brown, J. B.; Cannon, B.; Lischer, C. E.; Davis, B.; Moore, E., and Murray, J.: Further Reports on the Use of Composite Free Grafts of Skin and Cartilage from the Ear. *Plast. and Reconst. Surg.*, 1-2:130-134, 1946.

34. Brown, J. B.; Cannon, B.; Lischer, C. E., and Davis, W. B.: Composite Free Grafts of Skin and Cartilage from the Ear. *Jour. A.M.A.*, 134:1295-1296, 1947.

35. Wachsberger, A.: Successful Autotransplantation. *Arch. Otolaryngol.*, 46:549, 1947.

36. Szlazak, J.: Repair of Nasal Defects with Free Auricular Grafts. *Brit. Jour. Plast. Surg.*, 1:176-180, 1949.

37. Converse, J. M.: Reconstruction of Nasolabial Areas by Composite Grafts from the Concha. *Plast. and Reconst. Surg.*, 5:247-252, 1950.

38. Dupertius, S. M.: Growth of Young Human Autogenous Cartilages. *Plast. and Reconst. Surg.*, 5:486-493, 1950.

39. Duformental, C.: Repair of Ala Nasi with Free Graft from the Ear. *Jour. de Chirurgie*, 67:485-490, 1951.

40. Peer, L. A., and Walker, J. C.: The Behavior of Autogenous Human Tissue Grafts. *Plast. and Reconst. Surg.*, 7:6-23, 73-84, 1951.

41. Farina, R.: Loss of Substance from the Nasal Alae and Tip. *Plast. and Reconst. Surg.*, 9:52-54, 1952.

42. Maximow, A., and Bloom, W.: "Textbook of Histology," p. 110. W. B. Saunders, 1952.

43. Laskin, D. M., and Sarnat, B. G.: Respiration and Anaerobic Glycolysis of Transplanted Cartilage. *Proc. Soc. Exp. Biol. and Med.*, 79:474-476, 1952.

44. Laskin, D. M., and Sarnat, B. G.: The Metabolism of Fresh Transplanted and Preserved Cartilage. *Surg., Gynec. and Obst.*, 96:493-499, 1953.

45. Laskin, D. M., and Sarnat, B. G.: Collective Review, Cartilage and Cartilage Implants. *Surg., Gynec. and Obst.*, 99:521-531, 1954.

46. Longmire, W. P.; Cannon, J., and Weber, R.: "General Surgical

Problems of Tissue Transplantation, Preservation and Transplantation of Normal Tissue," p. 23. J. A. Churchill, Ltd., 1954.

47. Billingham, R. E.: "Biological Applications of Freezing and Drying," p. 253. Academic Press, Inc., New York, 1954.

48. Peer, L. A.: Cartilage Grafting. *Brit. Jour. Plast. Surg.*, 7:250-262, 1954.

49. McLaughlin, C. R.: Composite Ear Grafts and Their Blood Supply. *Brit. Jour. Plast. Surg.*, 7:274-278, 1954.

50. Rao, K. V. S.: Experimental Study of Regeneration in Cartilage. *Jour. Path. and Bact.*, 67:455-459, 1954.

51. Gibson, A.: Hyaline Cartilage, Degeneration and Regeneration. *Canad. Med. Assoc. Jour.*, 73:442-447, 1955.

52. Craigmyle, M. B. L.: Studies of Cartilage Autografts and Homografts in the Rabbit. *Brit. Jour. Plast. Surg.*, 8:93-100, 1955.

53. Peer, L. A.: "Transplantation of Tissue," p. 73-89. Williams & Wilkins, Baltimore, 1955.

PAN-PACIFIC SURGICAL ASSOCIATION.

The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 28 through October 5, in 1960.

All members of the profession are cordially invited to attend and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities.

An outstanding scientific program by leading surgeons promises to be of interest to all doctors. Nine surgical specialty sections are held simultaneously.

Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Director General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

HAMARTOMA OF THE TRACHEA.

Report of a Case, with a Review of the Literature of Benign Tracheal Neoplasms.

CHAS. F. ENGELKING, M.D.,

San Angelo, Tex.

The trachea, which is the segment of the respiratory pathway between the larynx and the main bronchi of the lung, is distinguished from these structures in a number of ways, most notably by its peculiar immunity to tumor formation. The incidence of bronchogenic malignant tumors has increased enormously in the past several years, but no similar increase has been noted in the incidence of tumors of the trachea. The number of tracheal tumors on record in the literature is, in fact, so limited that reports of individual cases of the rarer neoplasms are still justified.

CASE REPORT.

A 51-year-old white man was first seen Feb. 3, 1951, with the chief complaint of asthma. This diagnosis, he stated, had been made by a number of physicians whom he had previously consulted.

The patient had what he described as "throat trouble" off and on for the past 20 years, and for the last several years he had had progressively increasing difficulty in breathing, more marked when he had a cold. He also had some shortness of breath on exertion, and for some time had been unable to sleep recumbent. He had a severe cough, which was more pronounced in the recumbent position, and a moderate amount of mucopurulent sputum, which at times seemd to contain considerable pus but which had never been bloody. There was no pain in the chest, except after severe coughing spells, and no history of loss of weight.

When the patient was first seen, his breathing was noisy and obviously difficult. Examination of the chest, however, failed to disclose asthmatic sounds, and the findings were much more suggestive of obstruction somewhere in the trachea or in one of the bronchi than of asthma.

Examination of the ears, nose and throat was negative. There was normal movement of both the true and the false cords on phonation and respiration.

Although breathing was labored, chest expansion was normal bilaterally. Crepitant rales were heard over the lower right side of the chest. Elsewhere, the breath sounds were normal except that they were somewhat noisy because of an apparent mechanical obstruction in the trachea or the bronchi.

Editor's Note: This manuscript received at The Laryngoscope Office and accepted for publication Feb. 15, 1959.

Routine laboratory examinations were essentially negative.

A tentative diagnosis was made of bronchiectasis and neoplasm of the right main stem bronchus at the terminal end.

Roentgenologic examination of the chest Feb. 4, 1951, showed a rather marked, patchy emphysema throughout both lung fields. There was an area of increased density in the right cardiophrenic angle which the radiologist considered due to a bronchiectasis, though he noted that the presence of a rather smooth area of increased density in the hilar portion of this lung made it necessary also to consider the possibility of a malignant tumor.

Examination of the larynx and trachea Feb. 9, with air as a contrast medium, showed a definite narrowing of the trachea just below its opening from the larynx. The impression was that some type of mass was present on the posterior wall of the trachea, and direct visualization of the area was suggested as likely to be more useful than films.

Examination of the esophagus with barium Feb. 14 showed it to be normal in all views.

Feb. 7, the 9 mm. Jackson bronchoscope was easily passed for a distance of 21 cm. from the upper incisor teeth. At this point, a hard, polypoid, pedunculated, broad-based lesion was observed on the right posterolateral wall. Upon expiration, the lesion practically occluded the entire lumen of the trachea. On inspiration, it swung down against the tracheal wall, and there was no respiratory difficulty as there was on expiration. A specimen was secured from a suspicious looking, granulating area in the lesion; there was no bleeding.

When the main stem bronchus was entered, the mucous membrane was found to be extremely edematous and hyperemic, and a large amount of purulent material was aspirated, but no tumor was noted. The left main stem bronchus revealed hyperemia of the mucous membrane and a moderate amount of purulent material.

The prebronchoscopic clinical diagnosis of bronchogenic carcinoma and bronchiectasis was changed to fibroadenoma of the trachea and right bronchiectasis.

The pathologic report and histologic diagnosis was chronic bronchitis; probable squamous metaplasia of the trachea; bronchial exudate with squamous cells, but no evidence of any malignant change. The findings were regarded as inconclusive and a second biopsy was recommended.

Bronchoscopy was repeated Feb. 12, 1951. The bronchoscope was passed until the tumor mass previously described was encountered. It extended downward for several tracheal rings and practically obstructed the entire tracheal lumen except for the anterior portion. The postbronchoscopic diagnosis was again adenoma of the trachea. The specimens which were secured with only minimal bleeding were reported by the pathologist as one of the very uncommon benign fibromas of the trachea. It was not possible to determine from the specimens whether the tumor was uniformly fibrous throughout or might contain mixed elements, or even cartilage.

During his first few days in the hospital, the patient continued to cough and to present all the signs of a respiratory obstruction. He could not lie down in bed and had to sleep sitting up, though with medication, it was possible most of the time to secure adequate rest and sleep for him.

Operation was performed Feb. 15, 1951, under general anesthesia administered by the endotracheal technique, on the diagnosis of fibroma of the trachea.

A midline tracheal incision was made from the thyroid cartilage over the cricoid cartilage and continued down in the midline of the neck to the sternal notch. After the isthmus of the thyroid was divided in the center, the second, third and fourth tracheal rings were then incised.

Examination of the trachea up to the glottic chink showed no abnormalities. The distal portion was occluded by the neoplasm demonstrated on bronchoscopy.

After the pleura was reflected from the trachea and the fifth, sixth, seventh, eighth and ninth tracheal rings were incised in the midline, the neoplasm was brought into view on the posterior right lateral wall of the trachea. It was circular, nodular and broad-based. The color was grossly that of the normal tracheal mucosa.

The mass was grasped with a tenaculum and with the tonsil snare wire but had to be removed by morcellation because the tonsil wire could not be placed over it in its entirety. It was excised down to the level of normal mucosa of the trachea. After the mass had been removed, the trachea was entirely patent, and the right main stem bronchus was also patent and unobstructed.

The tracheal rings were closed with chromic 0 catgut. A cigarette drain was placed into the superior mediastinum and the wound closed in the usual fashion. After a No. 6 tracheotomy tube, of specially long construction, had been inserted into the trachea, the skin edges were approximated with 0 silk.

The postoperative diagnosis was fibroma versus hamartoma of the trachea. The pathologic report revealed the first specimen measured 1.5 cm. in length while the second specimen, which consisted of the bulk of the tumor, weighed 6 grams.

The findings most closely suggested hamartoma of the trachea, and this diagnosis, which at first tentative, was also the pathologist's final conclusion, on the ground that the tumor was made up of superfluous normal tissue, and that more than one type of tissue was present.

The slides in this case were sent to four other pathologists, of national reputation in Texas and elsewhere. Two agreed with the diagnosis of hamartoma. The third, while agreeing that the tumor was benign, considered it a lipoma that had incorporated mucous and serous glands and smooth muscle of the trachea. The fourth also considered it a benign lipoma, on the ground that the glands and some other elements in it developed in the fat as the tumor protruded in polypoid fashion into the lumen. He added, however, that he would be the first to agree that the large amount of glandular tissue within the tumor was not easy to explain on the basis of simple incarceration in the course of extension of a lipoma into the tracheal lumen.

The patient was in excellent condition at the end of the operation and breathed fairly well while still in the reclining position. The usual postoperative routine was instituted.

Emphysema of the neck appeared on the first postoperative day and extended to the occipital areas and down to the clavicle; it began to disappear within 48 hours. The patient was up and about on the third postoperative day. When the tracheotomy tube was removed on the fifth day, he breathed satisfactorily, with no evidence of the previous obstruction. He stated that he had not felt so well in the past five years, and that this was the first time in that whole period that he had been able to sleep recumbent and really enjoy a night's rest. The sutures were removed on the sixth postoperative day. The patient was discharged from the hospital March 2.

The patient was observed at weekly intervals for the next three weeks. He had no complaints except for some cough and continued foul sputum, attributable to bronchiectasis. He returned to work April 1, and when he was examined April 14, he was in excellent condition and had no complaints. When he was re-admitted to the hospital June 9 for a routine postoperative check-up, he had no complaints of any kind. Bronchoscopy showed no evidence of a residual tumor.

The patient was observed at irregular intervals for four years after operation. On his last examination, in June of 1955, he was 30 pounds heavier than when he was first seen. He had no difficulties in breathing and no wheezing, regardless of his position, and he slept recumbent in complete comfort.

FREQUENCY AND CLASSIFICATION.

Tumors primary in the trachea, if not actually rare, are extremely infrequent in comparison with tumors of the larynx and bronchi.

Holinger and his associates[7] report the frequency of tumors of the trachea as compared with tumors of the larynx ranges from 1:300 to 1:800. According to Broyles,[2] tumors of the lungs and trachea are five times more frequent in males than in females and are most often observed between the fourth and sixth decades.

The collection of tracheal tumors recorded in the literature has grown slowly. At the time of D'Aunoy and Zoeller's[4] comprehensive report in 1929, all recorded primary tracheal tumors numbered only 351.

Benign tumors of the trachea, although infrequent, encompass a wide variety of pathologic types. In D'Aunoy and Zoeller's[4] collected material, there were chondromas, osteomas, papillomas, fibromas, intratracheal goiters, adenoma and cylindroma.

A comprehensive search of the literature has revealed no previous recorded instance of hamartoma of the trachea. None of the five pathologists who examined the slides of the tumor reported in this communication had this type of tumor in their collection of pathologic material. An inquiry of the Armed Forces Institute of Pathology produced the information that the single case in the files was an incidental finding at autopsy.[11]

The differing points of view of several excellent pathol-

ogists in the case recorded herewith are apparently entirely typical of tumors of the trachea, which seem to present more pathologic than clinical difficulties. According to Jackson and Jackson,[9] whose remarks were made in connection with adenomas of the trachea, if sections of these tumors should be submitted to various pathologists, some sections would still be classified as adenomas, but others would be reclassified as inflammatory tissue. The differences of opinion, continued these observers, would be no criticism of the histologists, but would, on the contrary, simply emphasize the difficulties of histologic diagnosis and the importance of the inflammatory processes that result from bronchial or tracheal obstruction.

Smith's[12] report of an adenoma is an excellent illustration of how sound pathologists may disagree about these tumors. The tumor which was removed by the external approach, from the trachea, was diagnosed by his own pathologist as a benign tumor of the bronchial type. When, however, the specimen was examined by five other experienced pathologists, it was variously diagnosed as: *1.* a benign bronchial adenoma, likely to recur; *2.* adenocarcinoma; *3.* adenocarcinoma arising from the minor salivary glands; *4.* a benign mixed tumor; and *5.* an aberrant tumor of the thyroid gland without evidence of malignancy. Whatever the correct histologic diagnosis might be, the condition was benign, for the patient, as in my own case, was alive and well at the end of five years.

Jackson[8] makes the excellent practical point that while histologically all tumors of the trachea can be placed in either the benign or the malignant group, some benign growths may, in effect, be malignant, in that they can kill the patient. Benign may be used in its conventional sense and for the sake of simplicity, but, he continues, a growth that causes death is not benign in the strictest meaning of the term. Death from asphyxia would certainly have occurred in my own case if the condition had not been relieved; the mechanical proof of that possibility was demonstrated at bronchoscopy.

PATHOLOGIC PROCESS AND PATHOLOGIC PHYSIOLOGY.

As in this case, tumors of the trachea are most frequently located on the posterior tracheal wall, which is far richer

in glandular tissue.[4] These tumors are most frequently located in the lower third and most infrequently in the middle third.[5]

The etiology of benign tracheal tumors is unknown, though in most of them it is evident that inflammation, if it is not the primary factor, is an important secondary factor. The inflammatory hyperplasia evident in the reported case is entirely typical.

From the standpoint of origin, tracheal tumors are classified as endotracheal, murotracheal and peritracheal. From the standpoint of location, they are classified as cervical and intrathoracic. From the mechanical standpoint, they are classified as obstructive and non-obstructive.

Practically all the symptoms and signs to which tumors of the trachea give rise can be explained on a mechanical basis.

Dyspnea is usually the earliest and most prominent symptom. It may be constant or paroxysmal, but is almost invariably worse at night. Cough, which at first is dry and nonproductive, becomes productive later as the tumor increases in size. Other symptoms include cough, fever, pain, hemoptysis, and expectoration, which is usually purulent and blood-tinged. Clubbing of the fingers and toes is sometimes present, and abscess of the lung has occurred.

Endotracheal tumors, as well as murotracheal and peritracheal growths, if their growth is not interrupted, will eventually obstruct the tracheal lumen because of their bulk or inward pressure.

DIAGNOSIS.

Wheezing, which is the outstanding symptom in bronchial tumors, may also be present in tracheal tumors. Jackson,[5] in fact, considers that a wheeze heard at the open mouth which he calls the asthmatoid wheeze, is the most important of all symptoms. It is best heard near the end of a forced expiration and is characteristically increased by exertion.

On the other hand, as Jackson has also correctly pointed out, everything that causes a wheeze is not asthma. My own

case well illustrates this point; the patient was treated for asthma for five years. Obstruction of the trachea causes a clinicopathologic syndrome which so closely resembles true asthma that every instance of clinical asthma should be studied for evidence of tracheobronchial disease. In Jackson's[5] own series, every tracheal or bronchial tumor which did not cause external bulging was mistaken for asthma because the patient had a wheeze. Since tumors of the trachea frequently grow slowly, a tracheal tumor may be present for years before its true nature is recognized.

A tentative diagnosis of tracheal tumor can be made from the history, physical examination and radiologic studies, but the presence or absence of a tracheal tumor, like the presence or absence of a bronchial tumor, can be confirmed or ruled out only by bronchoscopy. Examination under the fluoroscope with lipiodol, followed by roentgenologic examination, is of supplemental usefulness, but bronchoscopy followed by biopsy is the only conclusive diagnostic method.

In infants and children, the differential diagnosis of tracheal tumors includes laryngismus, stridulus and papilloma of the larynx. In adults, chronic tracheitis and chronic bronchitis may cloud the issue, but the great pitfall, as in the reported case, is asthma.

TREATMENT.

The diagnosis of a tumor of the trachea serves, *per se*, as an indication for its removal, since, as in my own case, even a benign tumor may so encroach upon the airway as eventually to cause serious symptoms.

If the tumor is small, it can usually be removed by bronchoscopic methods, either *in toto* or by morcellation. Jackson[5] warns against attempted control by radiotherapy, because of the extensive dosage required and the possible risk of subsequent perichondritis and chondronecrosis.

If, as in the case of hamartoma, the tumor is too large to be removed through the bronchoscope, it will be necessary to use the external approach. Excision should extend for several millimeters beyond the evident limits of the tumor.

If the excision is so extensive that closure of the trachea is impossible after the tumor is removed, some prosthetic device may be employed as recommended by Belsey[1] and by Clagett and his group.[2] This group utilized a polyethylene tube.

In summary, benign tumors of the trachea are extremely infrequent. To date, less than 550 have been reported. A wide variety of pathologic types is represented in the reported cases, but pathologists are frequently in disagreement concerning the histologic diagnosis. Classification into benign and malignant types is always possible, but a benign neoplasm may have malignant potentialities, in that, if not removed surgically, it may kill the patient by completely blocking the airway.

All benign tumors of the trachea, regardless of pathologic type, present much the same symptoms and signs, practically all of which may be explained on a mechanical basis. Diagnosis may be suspected clinically and roentgenologic examination is of supplemental usefulness, but a precise diagnosis is possible only by bronchoscopy and biopsy. Bronchial asthma is the chief diagnostic pitfall; the possibility of a tracheal tumor must be borne in mind in very case in which an asthmatoid wheeze is present.

All tracheal tumors must be removed; their mere presence is an indication for their excision by bronchoscopy if they are small enough to permit this technique, otherwise by intratracheal surgery.

The case of hamartoma reported in his communication bears out practically all of these generalizations. It is apparently the first case of the kind to be recorded in the literature.

REFERENCES.

1. Belsey, R.: Resection and Reconstruction of the Intrathoracic Trachea. *Brit. Jour. Surg.*, 38:200-205, Oct., 1950.

2. Broyles, E. N.: Bronchoscopic Experiences with Tumors of the Lower Respiratory Tract. *Ann. Otol., Rhinol. and Laryngol.*, 57:129-133, March, 1948.

3. Clagett, O. T.; Grindley, J. H., and Moersch, R. J.: Resection of the Trachea: An Experimental Study and Report of a Case. *Arch. Surg.*, 57:253-266, Aug., 1948.

4. D'Aunoy, R., and Zoeller, A.: Primary Tumors of the Trachea:

Report of Case and Review of Literature. *Arch. Path.*, 11:589-600, April, 1931.

5. GILBERT, J. G., and others: Primary Tracheal Tumors in Infants and Adults. *Arch. Otolaryngol.*, 58:1-9, July, 1953.

6. GREEN, M. A.: Masked and Pseudo-asthma. *Ann. Allergy*, 10:245-252, May-June, 1952.

7. HOLINGER, P. H.; NOVAK, F. J., and JOHNSTON, K. C.: Tumors of the Trachea. THE LARYNGOSCOPE, 60:1086-1109, Nov., 1950.

8. JACKSON, C.: Tumors of the Trachea. *S. Surg.*, 5:262-276, Aug., 1936.

9. JACKSON, C., and JACKSON, C. L.: Benign Tumors of the Trachea and Bronchi with Especial Reference to Tumor-like Formation of Inflammatory Origin. *Jour. A.M.A.*, 99:1747-1754, Nov., 1932.

10. NORRIS, C. M.: Tracheal Obstruction. THE LARYNGOSCOPE, 69:595-620, June 1949.

11. SILLIPHANT, W. M.: Personal communication.

12. SMITH, M. T.; Adenoma of the Trachea. *Arch. Otolaryngol.*, 43:405-407, Sept., 1947.

SECOND WESTERN REGIONAL MEETING OF THE INTERNATIONAL COLLEGE OF SURGEONS.

The second Western regional meeting of the International College of Surgeons is to be held in Las Vegas this November. Registration will be Sunday, November 22; Scientific Meeting, Monday and Tuesday, November 23 and 24, at the Stardust Hotel. Registration fee, $35.00—including banquet and cocktails. (There is no registration fee for interns and residents). Hotel reservations are to be made directly to the hotel. For further information concerning this meeting, contact Dr. F. M. Turnbull, Jr., 1930 Wilshire Blvd., Los Angeles 57, Calif.

CERVICAL SYMPATHETIC PARALYSIS.*†

WARREN E. WIESINGER, M.D.,

Oakland, Calif.

The cervical sympathetic pathway may be involved in a variety of lesions. These may be central, involving the brain or spinal cord, or peripheral, interrupting the fibers on their way to the organs supplied. In reviewing this subject, evidence will be presented that the pathway for the oculopupillary fibers is through the tympanum. Pathological entities which may produce cervical sympathetic paralysis will be discussed, along with tests for the localization of the level of the lesion in Horner's Syndrome. An awareness of the proximity of the sympathetic nerves and ganglia may serve to avoid their severance, or, if this is not possible, to warn the patient of the sequelae.

Interruption of the cervical sympathetic pathway results in miosis, ptosis, enophthalmos, anhidrosis and vascular hyperemia. This syndrome carries the name of Horner,[1] who described it in 1869. Prior descriptions have been credited to Claude Bernard, Remak and Weir, Mitchell, Morehouse and Keen[2]; nevertheless, Horner's name is tightly bound to the syndrome, and acceptance of the association is universal.

The miosis results from paralysis of the dilator pupillary muscle supplied by the post-ganglionic fibers from the superior cervical ganglion. Ptosis results from paralysis of the superior tarsal muscle of Müller. The enophthalmos is not fully understood; it is the opinion of many ophthalmologists that the slightly sunken appearance is due to drooping of the upper lid and is not a true enophthalmos. Others have ascribed it to paralysis of the orbital muscle of Müller, a band of smooth muscle in the floor of the orbit, whose contraction supposedly

---

*Submitted as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1958.

†I express my gratitude to Dr. Elizabeth Crosby, Professor of Neuroanatomy, University of Michigan, for her assistance in the experiment on the monkey.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Feb. 2, 1959.

causes a slight protrusion of the eyeball.[3] This muscle action is so slight that it is doubtful that its loss could account for the appearance of the eye as seen in Horner's Syndrome. The vasodilatation and dryness of the skin of the face are due to damage to the vasoconstrictor and sudomotor fibers which pass through the cervical trunk.

ANATOMY.

The pathway of the sympathetic outflow from the brain to the eye is a circuitous one, allowing opportunity for lesions in many locations to interrupt its action. Experimental work indicates that there is a pathway from the frontal lobe to the hypothalamus.[4] From here the fibers pass down the brain stem to end in the upper portion of the lateral sympathetic nucleus of the spinal cord. The preganglionic fibers originate here to end in the ganglia of the sympathetic trunks. The latter are two cords situated along the lateral aspect of the vertebrae, extending from the base of the skull to the coccyx.

The cervical portion of the trunk contains three ganglia, probably formed by the fusion of the originally segmental ganglia. The superior cervical ganglion is situated near the second and third vertebrae. It is the largest. The middle is near the sixth cervical vertebra. The inferior is situated near the seventh cervical vertebra and is frequently fused with the first thoracic ganglion to form the stellate ganglion. There is segmental arrangement in the thoracic, lumbar, and sacral regions. The preganglionic fibers, originating in the lateral sympathetic nucleus, are customarily described as passing by way of the white rami communicantes to the ganglia. Palumbo,[5] however, believes that the pupillo-ciliary fibers are an exception, because, in 95 per cent of his cases of upper thoracic sympathectomy, division of the first thoracic ramus and adjoining rami, and removal of the lower third of the stellate ganglion, did not result in Horner's Syndrome. It is his opinion that the pupillo-ciliary fibers reach the upper part of the stellate gangion by a separate paravertebral route. The cervical ganglia receive ascending fibers from the VIIIth cervical and upper thoracic nerves, most of the fibers going to the superior cervical ganglion. The post-ganglionic fibers

arising from the ganglia are distributed as grey rami communicantes to the adjacent cranial nerves (IXth, Xth, and XIIth), to the upper three or four cranial nerves, to the pharynx, and to the external and internal carotid arteries, around which they form plexuses. From these plexuses, fibers supply the dilator muscle of the iris, the smooth muscle portion of the levator palpebrae, the orbital muscle of Müller, the blood vessels, sweat glands and hairs of the head and face, and the lacrimal and salivary glands.

The pathway of the post-ganglionic fibers from the superior cervical ganglion to the pupil is usually described as leading from the plexus on the internal carotid artery, without deviation to the cavernous plexus. Kunz[6] states that the cells in the superior cervical ganglion send fibers via the internal carotid plexus, the ophthalmic, the nasociliary, and the long and short ciliary nerves to the radial muscle of the iris. Duke-Elder[7] describe the route as from the superior cervical ganglion to the carotid plexus, to the cavernous plexus; then to travel over the Gasserian ganglion along the first division of the Vth nerve, continuing with the nasociliary nerve and then with the long ciliary nerves, entering the globe with the long ciliary arteries (some, perhaps, running without a relay along the long and/or sympathetic root of the ciliary ganglion to the short ciliary nerves), traversing the epichoroidal space to reach the iris and terminate in the dilator muscle. Craig and Fuller,[8] and Bedrossian[9] are in agreement with Kunz and Duke-Elder.

A review of the literature reveals only one reference to a pathway of the oculo-pupillary fibers through the middle ear. Hubert,[10] in reporting a Horner's Syndrome following radical mastoidectomy, mentions that several filaments from the carotid sympathetic detach themselves and penetrate the middle ear cavity while traversing the anterior wall. He further states that they travel to the anterior part of the promontory underneath the mucous membrane, and sometimes in the depth of the bone of the anterior wall, to the petrosa. The sympathetic filaments coming from the carotid plexus and traversing the tympanum are then described as rejoining the plexus to reach the dilator muscle of the pupil finally by

accompanying branches of the long ciliary nerves. This pathway for the dilator fibers is considered rare by Hubert, for he states that the majority, and sometimes all, of the sympathetic ocular fibers continue along the carotid plexus without entering the middle ear cavity. Hubert, in his description of the branches which penetrate the petrosa from the middle ear, is obviously referring to the superior carotico-tympanic (small, deep petrosal) nerves. Morris[11] describes this connection of the tympanic plexus with the cavernous plexus through the superior carotico-tympanic branches, but makes no mention of function. Raeder,[12] in his discussion of the ramifications of the internal carotid plexus, referred to the probability of some fibers passing by way of the deep petrosal nerve to the sphenopalatine ganglion to innervate the orbital muscle, but did not assign pupillo-dilatation to this pathway.

By investigating the tympanic plexus (in the monkey), I was able to demonstrate the route of the pupillo-dilatory fibers from the superior cervical ganglion through the middle ear, via the superior carotico-tympanic branches. First, the superior cervical ganglion on the posterior aspect of the carotid sheath was identified. Stimulation of the ganglion resulted in dilatation of the pupil on that side. The ganglion was then cut and loss of dilatation of the pupil on that side was noted when painful stimuli were given. On the opposite side, the promontory of the middle ear was exposed through an endaural radical mastoidectomy approach. The mucoperiosteum of the promontory was scarified and the endaural incision closed. No effect was seen on the pupil. This procedure was repeated on a second monkey, and no effect was noticed. On a third monkey, after first ascertaining that normal pupillary responses were present, particular attention was given to the region just below the semi-canal and cochleariform process, but above and slightly anterior to the promontory, in the scarification. Immediately after recovery from anesthesia, it was apparent that a miosis was present. It is obvious from the photographs of the monkey, that ptosis and the appearance of enophthalmos is present, as well as miosis (see Figs. 1 and 2). The oculo-pupillary paralysis persisted. It was, therefore, demonstrated that the pathway of the dilator pupillary fibers is through the superior carotico-tympanic

Fig. 1. The picture above demonstrates appearance after interruption of the superior carotico-tympanic branches on the left side. Note the ptosis and appearance of enophthalmos in the left eye. Miosis is not well demonstrated, because the pupils are constricted due to the strong light.

branches, and that the location of the dilator pupillary fibers is high on the mesial wall of the middle ear.

It is conceded that the rarity of Horner's Syndrome following radical mastoidectomy militates against the superior carotico-tympanic pathway for the oculo-pupillary fibers, inasmuch as it would be expected that paralysis would often follow operations in this area; however, it must be emphasized that the nerves are situated in grooves in the bone and are, therefore, protected against injury during the usual careful removal of mucous membrane and granulation tissue. It

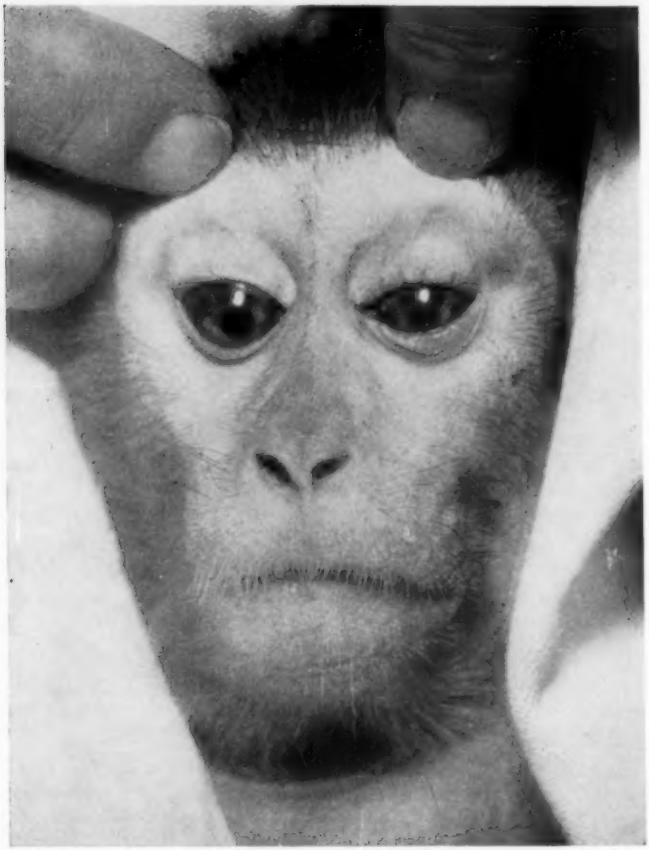

Fig. 2. A painful stimulus (pin-prick) is being applied. The right pupil is widely dilated as compared to the left. The superior carotico-tympanic fibers have been interrupted on the left side.

required actual notching of the bone with a sharp elevator to interrupt their continuity. This type of deep scarification is not part of the usual technique of radical mastoidectomy. The protection of the grooved bone in an area where rough instrumentation is hazardous can account for the "surgical immunity" of these branches; therefore, it is not necessary

to assume that the route, proven by the experiment in the monkey, is only an occasional one.

ETIOLOGY.

From the description of the neuro-anatomy involved, it is evident that a Horner's Syndrome may be produced by lesions in the brain, spinal cord, thorax, neck, and in the cranium.

Central lesions include thrombosis of the posterior inferior cerebellar artery, syringobulbia, syringomyelia, tumors of the cervical region of the spinal cord, and injuries in these regions. Kendall[13] emphasizes that the syndrome is not an isolated phenomenon, but a part of the complete neurological findings produced by these lesions.

Peripheral lesions involve pre- or post-ganglionic fibers; therefore, any lesion capable of producing interruption of these fibers, from the spinal cord to the termination of the post-ganglionic fiber, must be considered.

Egbert[14] reported a case following left lumbar paravertebral block at the level of L-I and L-II for relief of phlebitis in an 18-year-old female; 10 ml. of procaine was injected at each level. Spontaneous recovery was present in one-and-a-half hours. This case demonstrates the possibility of production of a Horner's Syndrome by diffusion of anesthetic solution through facial planes, even though the site of injection is remote.

DeJong[15] reported ten cases in which Horner's Syndrome was part of the findings on physical examination. The vulnerability of the sympathetic system to varying etiological factors in widely separated areas is well shown in his presentation. Horner's Syndrome was observed in a tumor of the spinal cord, syringomyelia, cervical pachymeningitis, aortic aneurysm, tumor of the upper part of the pulmonary sulcus, metastatic mammary carcinoma, and complicating thyroidectomy and phrenicotomy.

Intrathoracic lesions such as lymphosarcoma, Hodgkin's, bronchogenic carcinoma, esophageal carcinoma, and trau-

matic lesions such as gun shot wounds and knife wounds, may also produce the syndrome.

Pancoast[16] reported seven cases of a "superior sulcus" tumor in the upper part of the pulmonary sulcus in which Horner's Syndrome accompanied pain in the VIIIth cervical and first thoracic trunk distribution, and wasting of the muscles of the hand. On X-ray examination, this tumor showed a typically small, sharply defined shadow in the apex of the thorax with destruction of one or all of the upper three ribs and the adjacent transverse processes and, sometimes, slight vertebral body erosion. The specificity of this tumor has been doubted because the so-called "Pancoast Syndrome" is common to all sorts of lesions in this area.

In the neck, a large variety of lesions may be causative. Horner's Syndrome has been observed in traumatic wounds, thyroid adenoma,[17] in metastatic carcinomatous nodes and neurogenous tumors.[18] Weir Mitchell, Morehouse and Keen, in 1863, six years prior to Horner's description of the syndrome, gave a detailed observation of the clinical picture in a gun shot wound suffered by a Civil War soldier.[19] They correctly attributed the cause to the interruption of the cervical sympathetic nerves.

Operative procedures on the posterior pharyngeal wall in its lateral aspect may damage the internal carotid plexus, as shown in the following case:

G.S., aged ten, was seen on Nov. 29, 1952, because of difficulty in swallowing, of two months' duration. The past medical history was not relevant. There was no history of tuberculosis or other serious medical illnesses.

Examination revealed a smooth mass extending from the level just above the soft palate, downward to the hypopharynx and ending just above the level of the arytenoid cartilages. The main mass of the tumor was slightly more to the right of the midline. A chocolate colored fluid was removed by aspiration. This had the appearance of old blood (see Fig. 3).

Removal of the tumor was accomplished under general anesthesia. A midline incision was made through the posterior pharyngeal wall, and the tumor was removed by dissection, blunt and sharp. Dissection was slightly more difficult from the right upper lateral surface.

The pathological report read as follows: "The submitted specimen consists of a lobular, irregular shaped mass of tissue measuring 5x3x2.5 cm. in size. One pole appears hemorrhagic and, on section, there are several cysts ranging from 5 mm. to 2 cm. in diameter. These are devoid

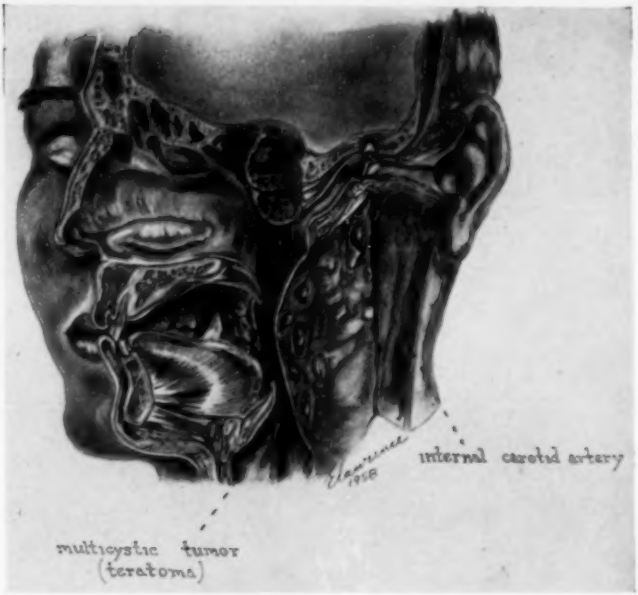

Fig. 3. The drawing is reproduced from Jackson and Jackson, "Diseases of the Nose, Throat and Ear," Fig. 231, page 287, published by W. B. Saunders Co., 1945, and is adapted to show teratoma, by E. Lawrence.

of content at the present time. The surrounding tissue is soft and spongy. The microscopic sections show multicystic lesions with the cysts lined by cuboidal, flattened and columnar epithelium. There are a few areas showing metaplasia. Scattered throughout are areas of lipoid granulation tissue with fatty acid crystals, old blood pigment, and proliferating fibroblasts. The main mass is composed of thymic tissue with characteristic thymic lobules, primary and secondary, and characteristic Hassell's Corpuscles. Diagnosis: cystic teratoma."

After a febrile postoperative period, the patient was discharged from the hospital. On the first postoperative office visit, a week later, the mother inquired as to why the left pupil was so large as compared to the right. Examination revealed paralytic miosis of the right eye.

The injury to the sympathetic pathway in this patient was undobtedly done in dissecting free the superior lateral pole of the tumor. The damage was to the superior cervical ganglion itself, none, I am thankful to say, to the internal carotid artery!

Johnson and Fisher[20] reported a case following intra-oral removal of a Schwannoma. They were able to find the superior cervical ganglion in the capsule of the tumor.

Further reminder of the proximity of the sympathetic pathway to the oral cavity has been given by Hald and Goldtfredsen[21] reporting on transitory Horner's Syndrome after paratonsillar anesthetization for local tonsillectomy. They reported 11 cases of transitory paralysis, two of which were bilateral.

Raeder[22] described involvement of the dilator pupillary fibers in the region of the Gasserian Ganglion. Because of the coincidence of oculo-pupillary paralysis and trigeminal symptoms the location was described as "paratrigeminal." His first patient with this syndrome had headache, vomiting, pain in the left trigeminal area, paresis of the left side of the palate, paresis of the pterygoid muscles, epiphora, miosis, and hypotonia on the left side without vasomotor or trophic disturbances. The patient died of tuberculosis, and postmortem examination revealed an endothelioma between the internal carotid artery and the Gasserian Ganglion. He reported four other cases; two of which were the results of head injuries with probable skull fracture, one due to probable herpes zoster, and one, assumed to be a tumor, as in his first case.

DIAGNOSIS OF THE LEVEL OF THE LESION.

Localization of the level of the lesion relies on the clinical evaluation of signs and symptoms, and on the use of drugs to test the level at which the pathway is interrupted.

Horner's Syndrome, in central lesions, is an incidental finding in the neurological picture produced by the lesion; localization will depend on the neurological findings accompanying the sympathetic paralysis. In peripheral lesions, absence of sweating (anhidrosis) places the lesion at some place proximal to the external carotid plexus. Conversely, in a Horner's Syndrome where sweating is present, the lesion is peripheral to where the external carotid plexus takes off from the main sympathetic trunk.

It is convenient to divide the neuro-anatomical pathway into

its component neurons for the purpose of description of the action of drugs on the pupil at different levels. The fiber from the hypothalamus to the cervical portion of the cord at the level of the VIIIth cervical and first thoracic is called the first neuron. The second neuron (pre-ganglionic) ascends in the neck through the inferior and middle ganglia to the superior cervical ganglion. The third neuron arises from the superior cervical ganglion and is distributed to the plexuses intracranially, as previously described.[23]

The drugs employed in the pharmaco-dynamic tests are the parasympathetic depressants (atropine, homatropine, and their relatives), the parasympathetic stimulants (pilocarpine principally), and the sympathetic stimulants (cocaine and adrenalin).[24]

The action of epinephrine and cocaine on the pupil aid considerably in placing the lesion in the neuron involved. Epinephrine has no effect on the normal pupil; however, if the post-ganglionic fibers are interrupted, there will be dilatation of the pupil on administration of epinephrine. This is explained by the fact that when smooth muscle is deprived of its nerve supply it becomes hypersensitive to stimuli which normally mediate its action;[25] thus, the dilator fibers become sensitized, and minimal amounts of epinephrine will evoke a maximal response. If the lesion is central (first neuron), or preganglionic (second neuron), there will be no dilatation with epinephrine; cocaine, on the other hand, causes dilatation in the normal pupil. In lesions of the second or third neuron it will have no effect, but, for an unexplained reason, it will cause dilatation in lesions of the first neuron. In an oculo-pupillary paralysis due to a thoracic or cervical lesion, or one along the internal carotid artery, the pupil does not dilate in response to cocaine.

Pilocarpine injected subcutaneously (5 mg.) will render the mitotic pupil more constricted. No sweating will occur on the affected side, while marked sweating will occur on the opposite side. Pilocarpine is a parasympathetic ending stimulant which produces sweating by acting on the cholinergic post-ganglionic fibers of the sympathetic.

DIFFERENTIAL DIAGNOSIS.

The small pupil of a Horner's Syndrome (paralytic miosis) may be differentiated from a spastic miosis by the use of atropine and eserine. In the presence of a dilator paralysis, atropine gives a relatively slight mydriasis; in a spastic miosis, atropine produces a considerable dilatation. Eserine, in paralytic miosis, produces a maximal miosis, since the sphincter is completely unopposed. In a spastic miosis it produces no effect as the sphincter is already in spasm.[26]

SUMMARY.

1. The neuro-anatomy of Horner's Syndrome has been reviewed.

2. Evidence has been presented that the pathway of the dilator pupillary fibers is through the superior carotico-tympanic branches.

3. Pathological entities producing Horner's Syndrome have been presented.

4. Localization of the level of Horner's Syndrome has been discussed.

REFERENCES.

1. Horner, F.: "Uber eine Form von Ptosis," p. 193-198, Klein Mbl. Augenheilk, 1869.

2. Kish, B.: American Discovery (of Horner's Syndrome). *Bull. Hist. Med.*, 25:284-288, May-June, 1951.

3. Strong, O., and Elwyn, A.: "Text Book of Neuro-anatomy," 3rd Ed., Chap. 12, p. 173. Williams & Wilkins Co., 1953.

4. Strong and Elwyn: Ibid, p. 288.

5. Palumbo, L. T.: New Surgical Approach for Upper Thoracic Sympathectomy. *Arch. of Surg.*, 76:807-810, May, 1958.

6. Kunz, A.: "Autonomic Nervous System," Chap. 20, p. 325. Lea and Febiger, 1945.

7. Duke-Elder, W. S.: "Text Book of Ophthalmology," Vol. IV, Chap. 44, p. 3736. The C. V. Mosby Co., St. Louis, 1929.

8. Craig, J. D., and Fuller, R. C.: Cervical Sympathetic Paralysis. *Brit. Med. Jour.*, 1:1182-1184, June 19, 1948.

9. Bedrossian, E. H.: Raeder Syndrome. *Arch. of Ophthalmol.*, 48:620-623, Nov., 1952.

10. Hubert, L.: Horner's Syndrome with Chronic Purulent Otitis Media: Demonstration of Diagnostic Test. The Laryngoscope, 53:46-49, Jan., 1943.

11. Morris: "Human Anatomy," ed. Schaeffer, J. Parsons, 11th Ed., p. 1197. The Blakiston Co., 1953.

12. Raeder, J. G.: "Paratrigeminal" Paralysis of Oculo-pupillary Sympathetic. *Brain*, 47:149-158, May, 1924.

13. Kendall, D.: Horner's Syndrome. *Practitioner*, 177:1062, Dec., 1956.

14. Egbert, L. D.: Complication of Lumbar Sympathetic Block. *Anesthesiology*, 16:811-812, Sept., 1955.

15. DeJong, R. N.: Horner's Syndrome; Report of Ten Cases. *Arch. of Neurol. and Psychiat.*, 34:734-743, Oct., 1935.

16. Pancoast, H. K.: Superior Sulcus Tumor. *Jour. A.M.A.*, 99:1391-1396, Oct. 22, 1932.

17. Blackwell, C. C.: Horner's Syndrome Associated with Goiter. *Mil. Surg.*, 95:219-222, Sept., 1955.

18. Conley, J.: Neurogenus Tumors in the Neck. *Arch. Otolaryngol.*, 1:167-180, Feb., 1955.

19. Kisch, B.: op. cit., May-June, 1951.

20. Johnson, F., and Fisher, R.: Schwannoma of the Pharynx with Horner's Syndrome. The Laryngoscope, 67:815-822, Aug., 1957.

21. Hald, E., and Godtfredsen, E.: Transitory Occurrence of Horner's Syndrome After Paratonsillar Anesthetization for Tonsillectomy. *Acta Oto-Laryngol.*, 30:156-161, 1942.

22. Raeder, J. G.: op. cit., May, 1924.

23. Jaffe, N. S.: Localization of Lesions Causing Horner's Syndrome. *Arch. of Ophthalmol.*, 44:710-728, Nov., 1950.

24. Duke-Elder: op. cit., p. 3739.

25. Stine, G., and Draper, P.: Horner's Syndrome. *Rocky Mount. Med. Jour.*, p. 504-507, July, 1945.

26. Duke-Elder, W. S.: op. cit., p. 3741.

3022 East Fourteenth St.

PYOCYANEOUS OSTEOMYELITIS OF THE TEMPORAL BONE, MANDIBLE AND ZYGOMA.*†

PHILIP E. MELTZER, M.D.,
and
GEORGE KELEMEN, M.D.,
Boston, Mass.

The distinct pathologic entity, osteomyelitis of the temporal bone, infrequent in occurrence before the advent of chemotherapy and antibiotics, has all but disappeared today. Even among the rare reports some must be weeded out, as they do not conform to the classic description first published by Toulmouche in 1838.

The following case belongs unmistakably under this heading and presented some unusual features:

Case History. (Massachusetts Eye and Ear Infirmary, 92-65-28). A 61-year-old man with severe diabetes was referred by Dr. Louis G. Lytton, of Miami, to Dr. Meltzer on May 3, 1956, with the following history:

The patient was first seen by his family physician because of pain in the right ear that developed in December, 1955, and grew worse. Local treatment to the ear failed to relieve the pain, so he was referred to the otologist, Dr. Lytton, on February 3, 1956. Examination revealed a markedly tender, edematous external auditory canal.

For the next 11 days the patient was returned to the care of his family physician, during which time he received Combiotic twice daily for six days as well as local treatment to the inflamed ear. The local swelling improved but the patient still complained of severe, right-sided pain at night that required a narcotic for relief.

On February 14, 1956, Dr. Lytton found the nose and throat to be normal. There was a pulsating, purulent discharge from the right external auditory canal with protrusion of granulation tissue. Culture of the pus revealed Bacillus pyocyaneus, reported to be sensitive to Chloromycetin and streptomycin.

Chloromycetin, in 250-milligram doses, was given every six hours for six days, without effect. Then two grams of dihydrostreptomycin were given daily for a week, also without effect. The symptoms of pain, discharge, and preauricular and postauricular swelling persisted for two months, during which time five biopsies of the granulation tissue were

*From the Department of Otolaryngology, Harvard Medical School, and the Department of Otolaryngology, the Massachusetts Eye and Ear Infirmary.

†Aided by United States Public Health Service Grant B-1272(C).

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 2, 1959.

reported to be pyogenic granulation tissue. Repeated blood counts showed a moderate increase of the white cells but nothing else of importance.

The blood sugar was difficult to control throughout this period, but the CO_2 combining power was within normal limits, and the urine, which occasionally revealed sugar, was always acetone and diacetic acid free.

Roentgenographic examinations of the mastoids, including the petrous pyramids, were taken on February 16 and on March 24, 1956, and there was no evidence of any disease. On April 4, destruction of the right mastoid was revealed for the first time on roentgenograms.

On April 5, a right radical mastoidectomy was performed by Dr. W. Hotchkiss, Dr. J. F. Hauss, and Dr. L. G. Lytton. Operation revealed marked softening of the mastoid-cell partitions but no gross destruction or free pus. The biopsy report was again "granulation tissue." Pyocyaneus was obtained from smears and cultures taken during the operation.

The postoperative course was uneventful, except that there was a persistent purulent discharge from the cavity. Because sensitivity tests indicated that the germ was sensitive to Chloromycetin, neomycin, and Furadantin, large doses of Chloromycetin were given intramuscularly from April 5 to April 14, without effect. The pain, swelling, and tenderness that extended over the right side of the face and over the zygoma were only temporarily relieved for the first five days after operation.

On April 14, the patient left the hospital against advice, but was readmitted on April 25 because of the intense pain over the right side of the head, zygoma, and face. A neurological consultant found no abnormalities. Roentgenograms failed to disclose any disease of the petrous tip or zygoma. The pyocyaneus was now sensitive to only neomycin and Furadantin, and therefore neomycin, in 2-gram doses daily for five days, and Furadantin, in 100-milligram doses four times daily for five days, were given, but without effect. The patient wished to return to his permanent home near Boston, so he was referred to Dr. Meltzer.

May 3, 1956. The examination revealed marked swelling on the right side of the face, with tenderness over the zygomatic arch. The mastoid cavity and the middle-ear space were completely obliterated by soft, pulpy granulations. Pressure on the right lateral portion of the face caused considerable oozing of pus from the operative cavity. The chief complaint was pain over the right side of the face and head. The patient was admitted to the Massachusetts Eye and Ear Infirmary.

May 4. Revision of right radical mastoidectomy. The infected granulations were completely removed from the mastoid and middle ear. The mastoid tegmen was removed, exposing a thin layer of granulation tissue over the dura. The peritubal cells, including the tensor tympani muscle, were thoroughly exenterated. No lead was found to account for the facial and zygomatic tenderness. There was no subtemporal infection. The posterior root of the zygomatic arch was explored, and no infection was found.

The revision failed to relieve the pain or to diminish the discharge.

Miss A. Mangiaracine, the bacteriologist, reported Bacillus pyocyaneous in pure culture, not sensitive to any of the antibiotics that were previously used.

May 15. An electroencephalogram was taken. The report was "moderately abnormal, right posterior temporal, parietal slowing."

May 16. Roentgenograms showed: "left mastoid, no destruction; right

mastoid postoperative; petrous negative." There was no evidence of osteomyelitis.

Right peripheral facial paralysis developed several days after the revision. Examination of the fundi showed retinal-vein engorgement. Because of the continuing headache, increased tendon jerk at the biceps and wrist on the right, and a focal electroencephalographic abnormality, a lumbar puncture was performed, which was normal.

June 5. Because pain and tenderness persisted over the zygoma and the right side of the face, and pus continued to well up in quantities that could not possibly arise from the mastoid cavity, reoperation was performed. An abscess deep in the infratemporal fossa was suspected.

Reoperation. An endaural incision was made, between the helix and the tragus, extending over the temporal muscle, granulations were cleaned

Fig. 1. Bone destruction and regeneration with cholesteatoma particles in the middle ear (hematoxylin-eosin, ×37).

from the middle ear, and the anterior canal wall was removed, exposing the fascia of the temporomandibular joint. A small fistulous lead was found, extending toward the zygomatic region. When the fistula was dilated, a gush of pus flowed from a deep abscess. A Miller-Abbott tube was inserted into the abscess cavity. The facial nerve was investigated and found to be intact. The peripheral paralysis was due to edema of the sheath, evidently the result of osteitis. The nerve was exposed and decompressed by slitting of the sheath.

June 9. A jugular-foramen syndrome developed, with paralysis of the right vocal cord, right palate, and right pharyngeal wall, necessitating the introduction of a feeding tube. Roentgenograms now revealed for the first time osteomyelitis of the right zygomatic bone and zygomatic arch.

June 11. Stereoroentgenograms revealed for the first time decalcifica-

tion of the right petrous apex, with soft-tissue swelling in the vault of the pharynx.

June 12. Neurological examination by Dr. P. R. Dodge revealed extensive involvement of the cranial nerves at the base of the skull, secondary to osteomyelitis and extradural infection, with the question of granuloma. The cerebrospinal-fluid findings on lumbar puncture with pressure on the jugular veins suggested thrombosis of the right internal jugular vein at the bulb or above, which, it was believed, would account for the electroencephalographic findings.

Dr. H. Blotner, the medical consultant, found the blood sugar, which occasionally rose to 315 mg. per 100 ml., extremely difficult to control, during the entire hospitalization.

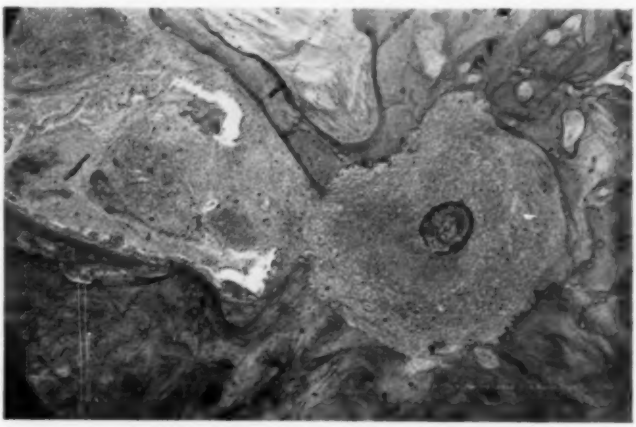

Fig. 2. Suppuration breaks open and fills a peritympanic pneumatic cell, destroying its walls (hematoxylin-eosin, ×37).

By June 14, because of the persistent pain and swelling, it was decided to remove the osteomyelitic bone and also the right ramus of the right mandible, including the condyle and coronoid process, in order to get direct treatment to the infected area in the region of the infratemporal fossa. This decision was made after consultation with Dr. G. W. Taylor for surgical opinion and Dr. E. H. Kass for the bacteriological aspects of the problem.

June 15. Dr. Taylor ligated the external carotid artery and removed the parotid gland. Dr. Meltzer removed the ascending ramus of the mandible, including the condyle and coronoid process, the zygomatic arch, and part of the zygomatic bone, exposing a huge abscess lying on the pterygoid muscles. The anterior and inferior external-auditory-canal walls were removed, exposing the jugular bulb. The petrous apex was entered by removing of the bone over the internal carotid artery as it traversed through the petrous apex. Pain was relieved after this extensive procedure.

The B. pyocyaneus was reported to be sensitive to polymyxin and neomycin by Dr. Kass's laboratory; therefore, iodoform gauze, soaked in polymyxin and neomycin in ¼ of 1 per cent lactic acid solution, was packed in the wound; this solution was changed three times a day. In spite of the constant saturation of the field with these antibiotic solutions, culture from what appeared to be a clean cavity revealed luxuriant growth of pyocyaneus. When these antibiotics were tested against the organism cultured at the Infirmary's laboratory by Miss Mangiaracine, however, they were completely ineffective.

Following the operation of June 15, although there was no new evidence of systemic or local infection, the patient's condition indicated that he

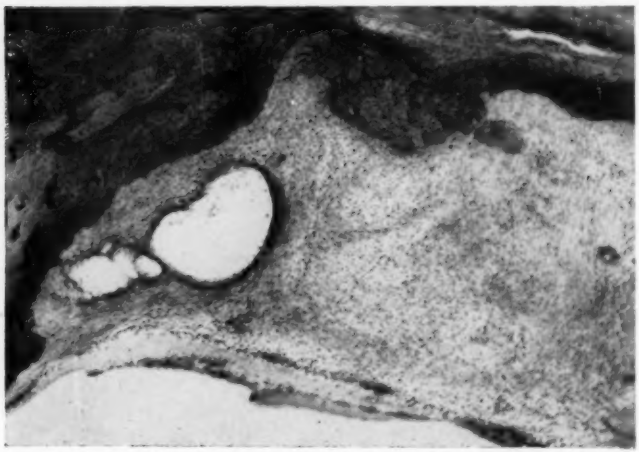

Fig. 3. Callus repair of a facial-canal perforation (hematoxylin-eosin. ×80).

was failing. The condition of the local wound, however, was good. The granulations appeared to be healthy in spite of the fact that there was an abundant growth of pure cutule of Bacillus pyocyaneus.

July 13. On removing of the packs from the wound, the internal carotid artery was pulsating wildly, and the arterial wall ballooned out and ruptured. The packing was immediately replaced. Under general anesthesia, an incision was made along the sternocleidomastoid muscle. The carotid sheath was exposed, and the common carotid artery was clamped and tied. The wound was examined, and a large hole in the carotid artery was found.

July 14. The patient was stuporous and could not be aroused. Because of the rales in the lungs, an oxygen tent was advised. Death occurred in the afternoon.

The *autopsy* revealed the following anatomic diagnoses: Bacillus pyocyaneus infection of the right external auditory meatus, middle ear, and temporal bone—the mastoid, petrous, and zygomatic elements; neuropathy

of the right vagus nerve and also of the right glossopharyngeal, accessory, and hypoglossal nerves, secondary to infection in the petrous bone; diabetes mellitus; thrombosis of the right internal jugular vein; aneurysm formation and rupture of the right internal carotid artery, in the extracranial portion; extradural hemorrhage, slight, over the petrous bone; bronchopneumonia, bilateral, severe; pleuritis, fibrous, obliterative, on the right side; and nephrosclerosis, slight.

The microscopical findings confirmed the fact that the cause for the multiple cranial-nerve palsies was to be found outside the brain stem.

PATHOLOGICAL INVESTIGATION OF THE EAR.

The entire *right petrous bone* was removed. After decalcifi-

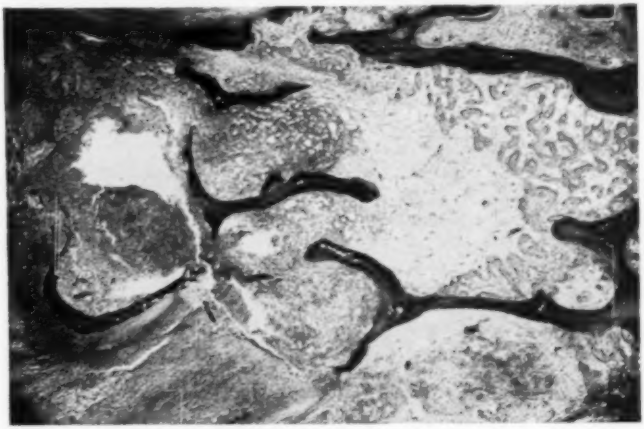

Fig. 4. Abscess and new trabecular bone in the petrous apex (Heidenhain-Mallory, ×37).

cation it was embedded in celloidin. Cutting on a horizontal plane, 750 serial sections were obtained and stained with hematoxylin-eosin and according to Heidenhain-Mallory. Unstained sections were mounted for observation under polarized light.

The bone that formed the medial wall of the *operative cavity* was found to be smooth. A few *mastoid cells* and a number of *perilabyrinthine cells* were present. The walls of the cells were jagged and were in the process of being reabsorbed. The mucous-membrane lining of the cell walls had been trans-

formed into high cushions of granulations that almost occluded the lumens of the cells. The bases of the cushions of granulations were lined with cysts in typical rosary formation. The vessels in the granulating tissue were engorged, but there was no cuffing. Wherever a lumen was still preserved it was filled with pus that contained diffuse cellular infiltrations or white blood cells in nests. A few cells contained clear transudate.

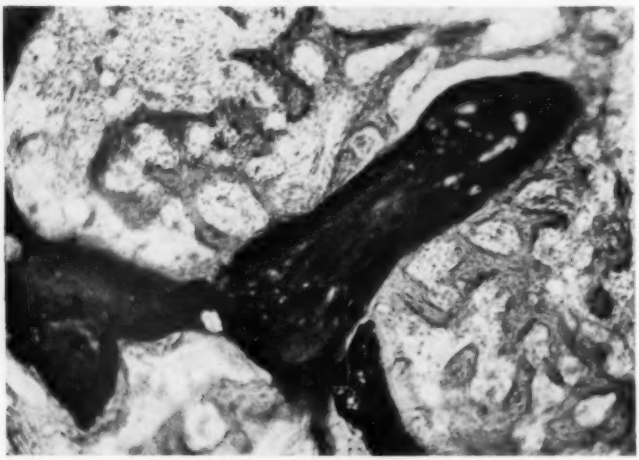

Fig. 5. Part of Fig. 4 at higher power (Heidenhain-Mallory, ×125).

The upper half of the lateral wall of the *facial canal* was missing, and had been replaced by gold foil, which was completely embedded on all sides in granulations and broken into several small particles that were divided into thinner sheets. The walls of the lower half of the facial canal were partly splintered, and an attempt to cover the smaller breaks by callus formation had started; the callus contained rare osteoblasts, and had not attained regular lamellar formation.

New bone was scattered throughout the remnants of the perilabyrinthine cells and was osteophytic (since there were no osteoblasts). *This* new bone had no relation to the new bone

that was formed to repair the destruction around the intercellular septa.

Masses of lamellae were embedded in the purulent content of the mastoid cavity, and were thought to be remnants of a *cholesteatoma.*

The *ossicles* were missing. The *oval window* was wide open, and only the lateral extremity of the annular ligament was preserved. The suppurative mass penetrated approximately up

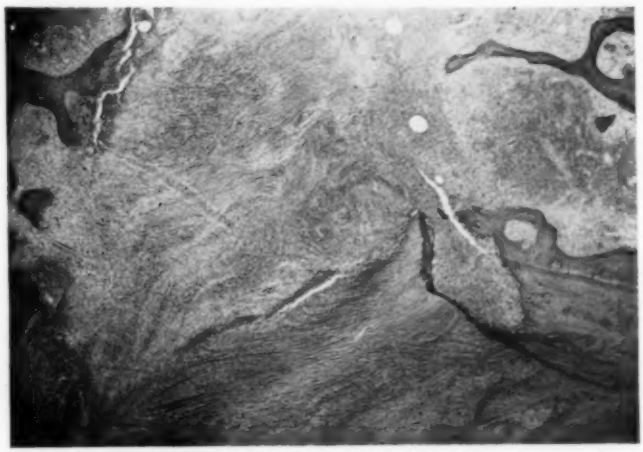

Fig. 6. Wide penetration of the apical abscess under the dura through the gap in the tegmen (hematoxylin, ×37).

to the level of what was originally the stapedial plate. High in the stapedial niche there was a break in the bony wall, and for its repair a plug of new bone had been formed. The niche of the *round window* contained some clumps of granulation tissue and blood. There was a blood clot on the cochlear aspect of the membrane of the round window.

The *vestibulum* was essentially free. The endolymphatic space contained only a few clumps of transudate. There was extravasation of perilymph in many points. There was hemorrhagic infiltration into the basal tissue of the cristae.

The *cochlea* was essentially free. Rosenthal's canal was well filled and contained blood in places. The spiral ligament contained blood in some spots and calcified vessels. Pigment was deposited in the vascular stria. The membrane of Reissner was straight in the basal turn and bulged slightly in the middle and apical turns. The organ of Corti was flattened, probably artificially. The basal turn contained some extravasate. The endosteal lining bridged clearly over the orifice of the cochlear aqueduct.

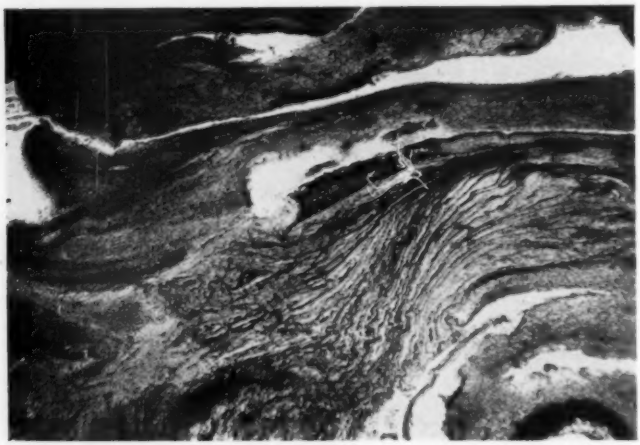

Fig. 7. Wide break in the carotid wall; thrombus in the arterial lumen, and thrombus covering, from the intracranial side, the break in the canal wall (hematoxylin-eosin, ×35).

The *inner acoustic meatus* contained an insignificant amount of blood, which was the only abnormality present within the meatus.

The *apex of the petrous bone* was basically diploic in cell structure in its entire extent. The septa were essentially intact, but the whole tip was transformed into a large abscess with a center of fibrous organization. The periphery of the abscess was a mass of free pus, which was formed among the broken-down trabeculae. The fibrous center of the abscess

showed a most intensive new-bone production. The trabeculae of the newly formed bone that originated from the septa showed active osteoblastic seams on many of their surfaces as evidence that further growth could have obliterated many of the cellular spaces of the original structure. A regular lamellar structure was not produced, and a lower calcium content was revealed by the conspicuous tinctorial difference. More regularity of the lamellar structure, however, was revealed under polarized light.

The apical portion of the abscess had a wide opening communicating under the dura, where it formed an *extradural abscess.*

The adventitia of the *carotid artery* contained large calcified plaques, and the lumen contained both free and thrombotized blood. A wide opening in the bony wall of the carotid canal led into the extradural space, through which its contents protruded under the dura. A thrombus covered the opening from the dural side.

The *dura* over the tegmen was very thick and cicatrized, and there were some areas of calcification. The *ganglion of Gasser* appeared to be intact.

Corpora amylacea were found in whole rows in the hiatus of the facial nerve, in the septa of the Gasserian ganglion, high up in the vestibular aqueduct, and were numerous in the internal acoustic meatus where the small vessels showed indications of being eventually transformed into psammomas.

DISCUSSION.

According to the classification of Brunner (1942, 1946, 1947) this case may be called an acute, protracted, progressive osteomyelitis. A detailed discussion of this condition was given lately (Kelemen, 1955 [rev. 1957]) so this comment will be limited to the outstanding or unusual findings of this particular case.

A characteristic leaping from focus to focus, with intact areas in between, indicating hematogenous spread, was present on both macroscopical and microscopical observation. Macro-

scopically, there was manifestation in the external, middle, and internal ear, under the temporal-bone squama, and in the zygoma. Invasion of the periosteomyelitic swelling in the region of the zygoma was considered by some authorities as one of the first signs of osteomyelitis. The labyrinth was entirely bypassed; the wide penetration through the oval window must have been of recent origin, as it did not reach beyond the level of the stapedial plate. Microscopically, between the perilabyrinthine cells with destroyed septa that were replaced by suppurating and granulating masses, other cells were found showing merely collateral, catarrhal signs.

The history did not contain anything to suggest a traumatic origin. Infection of the venous sinuses must have occurred late in the course of the disease. There are conflicting opinions in regard to the role of infection of the dural venous sinuses in osteomyelitis of the temporal bone; some reporters have maintained that sinus infection was the general rule, whereas others have maintained that it was very infrequent.

The term osteomyelitis may be applied when the disease of the spongiose substance is manifested both clinically and pathologically. Osteitis involves bony plates that do not contain marrow, whereas infection of the marrow is the most important feature in osteomyelitis. Osteomyelitis is very rare, notwithstanding the fact that there are temporal bones that never pneumatize and, therefore, remain especially prone to the infection. Anatomic conditions fail to explain why an inflammation in the middle ear is followed by a simple otitis in one case and in another by a septic process—an osteomyelitis.

In this illness the three outstanding features were the diabetes, the fact that the infecting organism was Bacillus pyocyaneus, and then the resultant pathological bone formation.

The *diabetes*, present before the ear infection, was always difficult to control, and in itself probably would have been quite capable of producing an ear complication. As insulin was of comparatively little avail, one is reminded of the pre-insulin era and its fearful "otite diabétique." In a discussion

of diabetes and the ear (Kelemen, 1955) it was emphasized that hemorrhage has always been a great danger in diabetes and ear disease. There can be no doubt that the course of the patient in this case was much influenced by the presence of the diabetes.

The problem of pyocyaneous infection in relation to the ear was discussed by Zaufal (1873). In a number of his patients with "blue aural discharge" the "Vibrio cyanagenus," or the "cylinderbacterium":"Bacterium termo," was isolated. Chambers (1900) tabulated 58 cases of otitis media in which the micro-organism was determined; in six of these cases pyocyaneus was found. According to his experience, if pyocyaneus alone is present, one half of the infections may be cured, with the others proving to be "stubborn." Wakefield (1904) obtained pure growth of pyocyaneus, at autopsy, from otogenic abscesses of the brain and of the cerebellum, as well as from the lungs and the heart.

This initial period came to a close with the monograph of Voss (1906) on the role of the pyocyaneus in aural disease. He analyzed 37 personal observations and added animal experiments. One of his patients (Case No. 17) was a man 25 years of age who had had a chronic otitis and intermittent discharge since early childhood. The phase of acute exacerbation lasted about six weeks and ended fatally. A subperiosteal and a retropharyngeal abscess and an abscess in the temporal lobe developed. The mandible was affected as well as the mandibular joint. Several operations were necessary. Certain aspects of this history show similarity with the presently discussed case; analogies would probably be even more numerous except that roentgenograms were lacking at that period and, consequently, conditions around the pyramid remained unrevealed. Even so, the evolution of the retropharyngeal abscess was traced, showing progression from the mandible along the tip of the pyramid.

After Voss elucidated the general role of pyocyaneus in otology, Haymann (1914) showed in guinea-pig experiments that artificial infection of the middle ear with pyocyaneus invariably proceeded to a diffuse, suppurative labyrinthitis. In a patient with otomastoiditis, Wirth (1926) isolated an-

hemolytic streptococci that acquired invasive character only after the pyocyaneus appeared; he was able to reproduce this sequence in mouse inoculations. Stanley (1947), in discussing pyocyanous infections, stated that pyocaneous bacteremia usually terminates fatally despite any kind of therapy. Because he found the organism in the pharynx he concluded that propagation to the ear was logical. In Stanley's cases of pyocaneous meningitis, six were preceded by otitis media, and although direct extension could not be demonstrated, there was bacteremia, and, therefore, he concluded that blood-borne metastases were responsible for the meningitis. Penicillin therapy inactivates the gram-positive organisms and thus apparently promotes the uninhibited growth of B. pyocyaneus and other gram-negative bacilli. Recently, the significance of this micro-organism was thoroughly discussed by Senturia (1957).

In the case under discussion, it is difficult to determine whether the strain of pyocyaneus was originally resistant to the numerous antibiotics administered, or if it developed resistance during the long course of the disease.

Even without the presence of the diabetes, the pyocyaneous alone would have been able to cause the fatal termination of the osteomyelitis. Stanley has described cases in which pyocyaneus sepsis occurred as a terminal complication in patients who were already suffering from other fatal diseases. In "Zinsser's Textbook of Bacteriology" (1948) it is stated that middle-ear infections, due to pyocyaneous can be extremely chronic and may persist for as long as five or ten years. In this case the rapid progress may be attributed to the presence of diabetes. Even with this general disease, diabetes, and the impossibility of any influence on it, the patient fought off for a considerable time the additional but inevitably fatal attack. According to the experience of this Department, pyocyaneus, which is so common in external-ear otitis, is frequently the cause of a very protracted middle-ear suppuration on which streptomycin or polymyxin may be of some influence. In the present case, however, these two drugs had no effect.

The *histological picture* showed a combination of both de-

struction and repair. In the middle ear there was a necrosis, which had the characteristics of the comparatively rare otitis media ossificans, with new bone composed partly of osteophytes and partly of the products of osteoblasts. In the tip the clear-cut picture of osteomyelitic new-bone formation was encountered. No other process is accompanied by any similar intensive attempt at repair to wall off a breach. The abscess in the pyramid progressed so that the center was fibrotic. Innumerable trabeculae of new bone originated from the old cell partitions; all were formed by osteoblastic carpets. No osteoid was present between the old and new bone since time was running short. The new bone, as was shown by its tinctorial properties, was low in calcium content, but observation under polarized light revealed its unmistakable lamellar character.

If there had been more time, the new trabeculae would have obliterated the formerly diploic cells of the tip completely. According to the roentgenographic findings, this condition in the tip could have been about one month old. Finally, by condensation of the central portion and isolation among the abscessed parts of the pyramid, the most characteristic product of osteomyelitis—the sequestrum—would have been created.

Although surrounded by the severest destruction on all sides, the inner ear still was not dead. Some hearing was preserved to the end, and dizziness was the evidence of a damaged but not extinguished vestibular organ; this was borne out by what was seen under the microscope.

Destruction of the apical tegmen was followed by the development of an extradural abscess, but the destruction of the bony wall of the carotid canal led to even more serious consequences. The contents of the canal, including the artery, now protruded through the break in the bony wall, forming an aneurysm. The arterial wall was so widely calcified that rupture would have been only a matter of time.

The final, dramatic incident was the hemorrhage from rupture of the carotid artery, for which two explanations may be postulated: namely, the changes found in the arterial wall

itself and the effect of the apical lesion. A voluminous body of literature offers ample discussion on both conditions.

The calcifications may have been present independently of and long before the final illness. In the media very extensive plaques were found that had accumulated most massively in the part of the artery that was nearest the cochlea. There was no progression in the plaques to bone formation.

According to Kecht (1937) the most frequent and dangerous forms of aneurysm formation in the carotid artery are those produced by erosion. In the case under discussion, dehiscences were seen in the bony wall of the carotid canal. It is possible that the carotid wall may have been very thin before the last impact and thus would have ruptured easily. Guild (1937) emphasized the fact that the carotid wall can be adherent to the wall of the canal; necrosis of the arterial wall can result from the adjacent suppurative process and thus result in hemorrhage. Similar topographic conditions induced Guild to advocate caution in the exenteration of the petrous tip if the access was along or through the carotid canal. Tato and von Soubirón (1935) and Podestá and Tato (1937) discussed the dangers of carotid hemorrhage in connection with the operation of Ramadier.

Mérei (1947) emphasized that although in children the intracanalicular part of the carotid artery contains strong internal and external elastic fibers, in adults the internal elastic lamina is well represented, whereas the external becomes less marked.

In the case under discussion, the propagation from the tip of the petrous portion of the temporal bone was spontaneous because exenteration of the apex of the petrous portion was not performed. The type of aneurysm encountered must be called "aneurysma spurium" because part of its wall was formed by the abscess in the apex. The aneurysm ruptured opposite the portion of the wall that was formed by the apical abscess, close to the most extended calcified plaques.

Ballance (1919) made the assertion that "every case of acute otitis which subsequently passes into chronic temporal bone disease has been wrongly treated at first." This state-

ment may be challenged, however, by this case in which prompt treatment was instituted from the very beginning but was not successful in averting a fatal outcome. Comparable cases have also been reported in which the most heroic surgical efforts were to no avail.

SUMMARY.

A fatal case of osteomyelitis of the temporal bone, mandible and zygoma is reported. During the eight months of the disease, B. pyocyaneus was constantly isolated from the secretions, and a variety of antibiotics were found to be of no help. The infection developed against the background of a severe and uncontrollable diabetes.

The surgical interventions to check the progress of the disease and the pathological findings in and around the temporal bone are discussed.

BIBLIOGRAPHY.

BALLANCE, C. A.: "Essays on the Surgery of the Temporal Bone," Vol. 1. Macmillan, London, 1919.

BRUNNER, H.: Pathologic Changes of Temporal Bone in Osteomyelitis of Skull. THE LARYNGOSCOPE, 52:954-967, Dec., 1942.

BRUNNER, H.: "Intracranial Complications of Ear, Nose and Throat Infections," Year Book Pub., Chicago, 1946.

CHAMBERS, T. R.: Bacteriological Examinations of Otitis Media Purulenta and Suppurative Mastoiditis. *Jour. A.M.A.*, 35:1405-1407, Dec. 1, 1900.

GUILD, S. R.: Hitherto Unrecognized Danger in Operation of Ramadier for Suppuration of Petrous Pyramid. *Acta Oto-laryngol.*, 25:6, 561-567, 1937.

HAYMANN, L.: Experimentelle Studien zur Pathologie der akutentzuendlichen Prozesse im Mittelohr (und im Labyrinth). *Arch. f. Ohrenheilk.*, 95:98-144, May, 1914.

KECHT, B.: Die Bedeutung der Arteria carotis interna in der Hals-Nasen-Ohrenheilkunde. *Arch. f. Ohren-Nasen, u. Kehlkopfheilk.*, 143:3-47, June, 1937; 144:2-52, Dec., 1937.

KELEMEN, G.: Aural Changes in Embryo of Diabetic Mother. *Arch. Otolaryngol.*, 62:357-369, Oct., 1955.

KELEMEN, G.: Osteomyelitis of the Temporal Bone. In "Otolaryngology." Edited by G. M. Coates, H. P. Schenck, and M. V. Miller. Hagerstown, Md., Prior, 1955. Chapter 20, pp. 26 (Revised in 1957).

MÉREI, J.: Contributions to Histology of Carotic Canal. *Oto-Rhino-Laryngol. danubiana*, 1:2-3, 122-127, 1947.

PODESTA, R., and TATO, J. M.: Histopathologischer Bericht ueber Einen Fall von Petrositis (Operation nach Ramadier). Riss und Blutung der

Carotis interna. Eiterige Hirnhautentzuendung. *Acta Oto-laryngol.*, 25: 254-261, May-June, 1937.

SENTURIA, B. H.: "Diseases of the External Ear," C. C. Thomas, Springfield, Ill., 1957.

STANLEY, M. M.: Bacillus Pyocyaneus Infections: Review: Report of Cases and Discussion of Newer Therapy Including Streptomycin. *Amer. Jour. Med.*, 2:253-277, March, 1947; 2:347-367, Apr., 1947.

TATO, J. M., and VON SOUBIRÓN, N.: Beitrag zur Frage der Pyramidenspitzeneiterungen. *Monatsschr. f. Ohrenheilk.*, 69:1454-1481, Dec., 1935.

TOULMOUCHE, M. A.: Observations d'Otorrhée Cérébrale; Suivis des Réflexions. *Gaz. Méd. de Paris*, 6:422-426, July 7, 1838.

VOSS, O.: Der Bacillus pyocyanaeus im Ohr. *Verocff. a. d. Geb. d. Militaer-Sanitaetswesens*, 33:34, 1-197, 1906.

WAKEFIELD, A.: Report of Fatal Case of Latent Temporo-sphenoidal Abscess of Otitic Origin, Followed by Multiple Secondary Cerebral Abscesses. *Arch. of Otology*, 33:273-282, July, 1904.

WIRTH, E.: Subakute Mastoiditis durch Mischinfektionen von Bacillus pyocyanaeus und Streptococcus anhaemolyticus. *Zeitschr. f. Hals-Nasen-Ohrenheilk.*, 17:2, 188-191, 1926.

ZAUFAL, E.: Ueber das Vorkommen blauer Otorrhoen. *Arch. f. Ohrenheilk.*, 6:2, 207-218, 1873.

"Zinsser's Textbook of Bacteriology," 9th Edition, Revised by D. T. Smith, et al. Appleton-Century-Crofts, New York, 1948.

A CLINICAL COMPARISON OF MONAURAL AND BINAURAL HEARING AIDS WORN BY PATIENTS WITH CONDUCTIVE OR PERCEPTIVE DEAFNESS.*

MARCEL BELZILE, M.D.,
Montreal, Canada,
and
DONALD M. MARKLE, M.Ed.,
New York, N. Y.

INTRODUCTION.

Although there exists an extensive literature dealing with monaural vs. binaural hearing, it is only recently that the use of binaural hearing aids have become a practical reality. Knudsen,[1] in 1939, Keys,[2] in 1946, Hirsh,[3,4] in 1950, and Broadbent,[5] in 1955, recommended the use of binaural hearing aids by hard-of-hearing individuals. Bergman,[6] in 1957, Carhart,[7,8] in 1958, and Markle and Aber,[9] in 1958, reported the successful use of binaural hearing aids by individuals with varying degrees and types of auditory impairment. It is the purpose of this report to present "A Clinical Comparison of Monaural and Binaural Hearing Aids Worn by Patients with Conductive or Perceptive Deafness."

SUBJECTS.

The subjects included in this study represent two selected groups of patients from the Hearing and Speech Center of New York University-Bellevue Medical Center. Fifteen subjects with bilateral conductive deafness and 15 subjects with bilateral perceptive deafness were investigated. All subjects had bilateral hearing losses with mid-three frequency (500, 1000, and 2000 cps) averages between 35 and 65 db.

In those cases with conductive deafness, the bone conduction

*Read before the Thirty-Fifth Annual Convention of the American Speech and Hearing Association, Nov. 17-19, 1958, New York, N. Y.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 18, 1959.

was not lower than 20 db at any one frequency, and the minimum "air-bone gap" was 25 db. In those cases with perceptive deafness, the difference between the air and bone conduction thresholds at any frequency was no more than 10 db.

The ages of the subjects varied between 16 and 70 years.

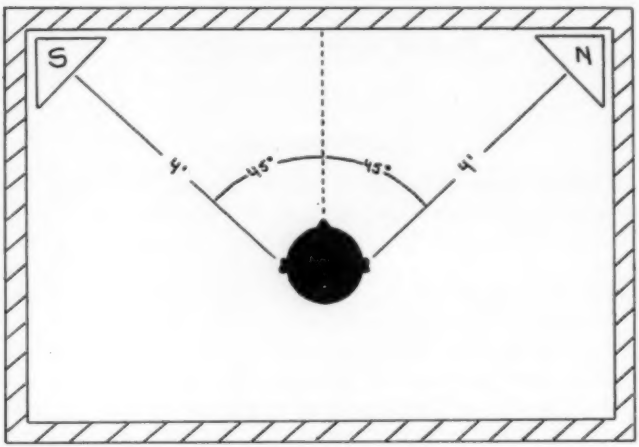

Fig. 1. Diagram of test situation illustrating position of subject in relation to signal (S) and noise (N) transducers.

PROCEDURE.

The subjects were seated in the center of the test room of a conventional two-room suite and, four feet away, and at a 45 degree angle to the left, was placed a loudspeaker (S) which is capable of delivering a signal at calibrated levels. Four feet away, and at a 45 degree angle to the right, was placed a second loudspeaker (N) capable of producing recorded noise at calibrated levels (see Fig. 1). Loudspeaker S is fed by an Allison 12-B hearing evaluation unit; loudspeaker N is fed by an Ideal T-1 auditory training unit.

In order to evaluate the relative values of monaural and binaural hearing aids, each subject was tested while wearing a conventional hearing aid in a conventional manner. In

addition, each subject was tested while wearing two conventional hearing aids—one mounted on each side of the head. In each case, the subject was instructed simply to set the volume of the instruments to a comfortable listening level for 50 db of recorded connected discourse (Fulton Lewis, Jr.).

The recorded test material consisted of phonetically balanced word lists (Auditory Test W-22). This material is prepared and distributed by Technisonic Studio.

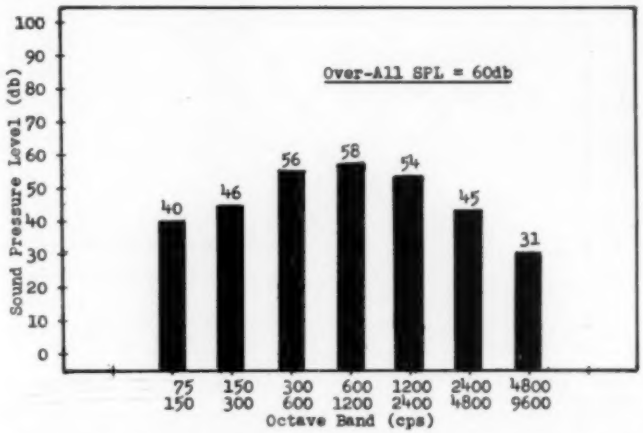

Fig. 2. Octave band)analysis of recorded factory background noise (Standard Sound Effect Record 300-A) as reproduced through an Ideal T-1 auditory training unit. This recording produces a constant acoustic output within ±1 db.

The noise used was recorded factory background noise (Standard Sound Effect Record 300-A) which is produced by Standard Radio Transcription Service, Inc.; this recording produces a constant acoustic output with ±1 db (see Fig. 2).

During the entire testing procedure, the noise was kept at a constant 60 db sound pressure level; this level was determined by measurements made with a General Radio 759-B sound level meter ("C" weighting network). In the presence of this noise, phonetically balanced word lists were presented

at levels of 30, 20, and 10 db above the noise, at the same intensity as the noise, and at 10, 20, and 30 db below the noise; this represents signal/noise ratios of +30, +20, +10, 0, –10, –20, and –30 db. The subjects' discrimination scores were obtained at each of these levels while wearing monaural and binaural hearing aids. The order of presentation was randomized for each subject.

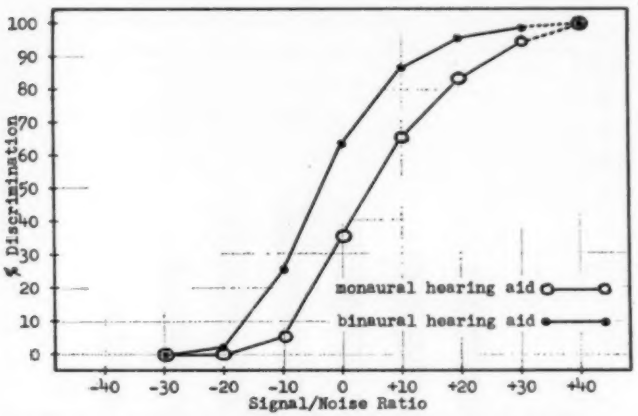

Fig. 3. A comparison of monaural and binaural hearing aids worn by 15 subjects with bilateral conductive deafness when discrimination for speech was determined at various signal/noise ratios. (Noise = 60 db SPL).

RESULTS.

Conductive Deafness.

As illustrated in Fig. 3, binaural hearing aids provide no significant advantage in auditory discrimination over monaural hearing aids when worn in relatively quiet surroundings (signal/noise ratios of +30 db or more) or in relatively noisy surroundings (signal/noise ratios of –20 db or less); the most significant improvement is demonstrated at signal/noise ratios between +20 and –10 db. At +20 db, the average improvement is 12.4 per cent with a range of 0 to 26; at +10 db, the average improvement is 20.9 per cent with a range of 8 to 52; at 0 signal/noise ratio, the average im-

provement is 27.4 per cent with a range of 2 to 58; at -10 db, the average improvement is 20.6 per cent with a range of 0 to 46 (see Table I). No patient demonstrated superior performance with the monaural over the binaural hearing aid at any signal/noise ratio.

Referring back to Fig. 3, and considering the 50 per cent discrimination level, it is noted that the average difference

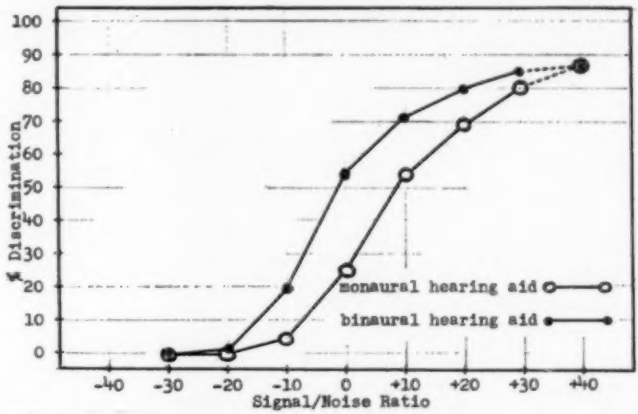

Fig. 4. A comparison of monaural and binaural hearing aids worn by 15 subjects with bilateral perceptive deafness when discrimination for speech was determined at various signal/noise ratios. (Noise = 60 db SPL).

in signal/noise ratios between monaural and binaural listening is approximately 10 db. In other words, while keeping the intensity of the signal constant, 50 per cent discrimination can be achieved in the presence of 10 db more noise while wearing a binaural hearing aid than can be achieved while wearing a conventional monaural instrument.

Perceptive Deafness.

As illustrated in Fig. 4, binaural hearing aids provide no significant advantage in auditory discrimination over monaural hearing aids when worn in the presence of signal/noise ratios of +30 db or more or -20 db or less; the most significant improvement is demonstrated at signal/noise ratios of

+10 and 0. At +10 db, the average improvement is 17.1 per cent with a range of 6 to 42; at 0 signal/noise ratio, the average improvement is 28.6 per cent with a range of 10 to 54 (see Table II).

At +30 db signal/noise ratio, one subject obtained 4 per cent better discrimination and four subjects obtained 2 per

TABLE I.

Discrimination Scores Obtained While Wearing Monaural and Binaural Hearing Aids in the Presence of 60 db SPL Recorded Factory Background Noise (N = 15 "Conductives").

Discrimination in Noise	Monaural		Binaural		Improvement	
	Mean	Range	Mean	Range	Mean	Range
S/N +30	95.2%	84-100%	99.7%	94-100%	4.5%	0-14%
S/N +20	84.1%	64-100%	96.5%	80-100%	12.4%	0-26%
S/N +10	66.4%	24-86%	87.3%	76-100%	20.9%	8-52%
S/N 0	35.8%	0-70%	63.2%	10-86%	27.4%	2-58%
S/N −10	6.6%	0-40%	27.2%	0-68%	20-6%	0-46%
S/N −20	0	-	2.0%	0-34%	2.0%	0-34%
S/N −30	0	-	0	-	0	-

TABLE II.

Discrimination Scores Obtained While Wearing Monaural and Binaural Hearing Aids in the Presence of 60 db SPL Recorded Factory Background Noise (N = 15 "Perceptives").

Discrimination in Noise	Monaural		Binaural		Improvement	
	Mean	Range	Mean	Range	Mean	Range
S/N +30	81.1%	56-100%	85.1%	58-100%	4.0%	−4-32%
S/N +20	69.4%	50-92%	80.2%	56-96%	10.8%	−4-28%
S/N +10	54.9%	14-72%	72.0%	56-88%	17.1%	6-42%
S/N 0	26.6%	0-52%	55.2%	26-82%	28.6%	10-54%
S/N −10	6.4%	0-22%	19.6%	0-64%	13.2%	0-44%
S/N −20	0	-	1.3%	0-20%	1.3%	0-20%
S/N −30	0	-	0	-	0	-

cent better discrimination while wearing a monaural hearing aid; at +20 db, one subject obtained 2 per cent improvement in auditory discrimination while wearing a monaural instrument. These differences are not considered to be significant.

Referring back to Fig. 4, and considering the 50 per cent discrimination level, it is noted that the average difference in signal/noise ratios between monaural and binaural listening is approximately 10 db. In other words, while keeping the intensity of the signal constant, 50 per cent discrimination

can be achieved in the presence of 10 db more noise while wearing a binaural hearing aid than can be achieved while wearing a conventional monaural instrument.

SUMMARY.

Within the structure of this clinical experiment, subjects with bilateral conductive deafness provide evidence of the superiority of binaural over monaural hearing aids when worn in listening conditions representing signal/noise ratios between +20 and –10 db. Subjects with bilateral perceptive deafness provide evidence of the superiority of binaural hearing aids when worn in listening conditions representing signal/noise ratios between +10 and 0. For both groups, the improvement is most evident at 0 signal–noise ratio.

For both the conductive and the perceptive deafness groups, 50 per cent discrimination can be achieved in the presence of 10 db more noise while wearing a binaural hearing aid than can be achieved while wearing a conventional monaural instrument.

REFERENCES.

1. KNUDSEN, V. O.: An Ear to the Future. *Jour. Acoust. Soc. Amer.*, 2:29-36, 1939.

2. KEYS, J. W.: "The Comparative Threshold Acuity of Monaural and Binaural Hearing for Pure Tone and Speech as Exhibited by 'Normal' and Hard of Hearing Subjects." Dissertation, Northwestern University, 1946; Abstract in *Speech Monogr.*, 14:203-204, 1947.

3. HIRSCH, I. J.: Binaural Hearing Aids; a Review of Some Experiments. *Jour. Speech, Hear. Disorders*, 15:114-123, 1950.

4. HIRSCH, I. J.: The Relation Between Localization and Intelligibility. *Jour. Acoust. Soc. Amer.*, 22:196-200, 1950.

5. BROADBENT, D. E.: Some Clinical Implication of Recent Experiments on the Psychology of Hearing. *Proc. Roy. Soc. Med.*, London, 48:961-968, 1955.

6. BERGMAN, M.: Binaural Hearing. *Arch. Otolaryngol.*, 66:572-578, 1957.

7. CARHART, R.: The Usefulness of the Binaural Hearing Aid. *Trans. Amer. Acad. Ophthal. and Otolaryngol.*, p. 120-126, 1958.

8. CARHART, R.: The Usefulness of the Binaural Hearing Aid. *Jour. Speech, Hear. Disorders*, 23:42-51, 1958.

9. MARKLE, D. M., and ABER, W.: A Clinical Evaluation of Monaural and Binaural Hearing Aids. *Arch. Otolaryngol.*, 67:606-608, 1958.

Notre Dame Hospital.
New York University Bellevue Medical Center.

THE ANTI-HEMORRHAGIC EFFECT OF ADRENOCHROME IN TONSILLECTOMY.

A Critical Evaluation.*†‡

ALEX WEISSKOPF, M.D.,

San Francisco, Calif.

Blood loss with surgery is a continuing spectre. Improved surgical technique, management of vascular collapse, blood chemistry and availability of blood for transfusion has markedly diminished surgical risk; nevertheless, excessive bleeding, difficult to control, is still a problem.

A wide variety of agents to control bleeding has been offered, ranging through the vitamins K and C, hormones, oxalic acid, calcium and, most recently, the adrenochrome derivative of epinephrine. All have been more or less enthusiastically received; however, controlled studies of surgical hemorrhage are noteworthy in their sparsity, for bleeding is most difficult to evaluate. How may one tell whether the bleeding stopped due to the use of a particular agent? Most bleeding fortunately stops spontaneously, or the practice of surgery would have "died aborning."

Nose and throat surgery is particularly fraught with the problem of bleeding, since so much of the work involves highly vascular areas which cannot be covered with skin or mucous membrane. Because of this, much of the literature and "detailing" of these agents has been directed toward ear, nose and throat and oral surgeons.

The literature on adrenochrome is most optimistic. Adrenochrome is an oxidation produce of epinephrine which is stabilized for clinical use as a semi-carbazone. This has been mar-

---

*The Adrenosem used in these studies was furnished through the courtesy of the S. E. Massengill Company, Bristol, Tenn..

†Read before the Forty-Third Annual Meeting of the Pacific Coast Oto-Ophthalmological Society, Las Vegas, Nev., May, 1959.

‡From the Division of Otorhinolaryngology, University of California School of Medicine, San Francisco, Calif.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication June 8, 1959.

keted as a salicylate under the trade name of Adrenosem.[3] It is reported that the hemostatic activity of epinephrine is also found in adrenochrome. Of particular interest is the absence of sympatheticomimetic activity. The mode of action is unknown. Animal experiments with Adrenosem[3][1,2] have shown a decreased extravasation of blood through the capillary walls of the hamster cheek pouch when injured with moccasin snake venom. It has been postulated that its mode of action is on the intercellular cement and ground substance about the capillary endothelium. There is no direct effect upon the vessel itself. Vasoconstrictive activity has not been demonstrated.

TABLE I.

Survey of the Literature on Adrenosem®.

Reference	Incidence of Bleeding Control	Adrenosem	Analysis
Bacala	19.8%	7%	No controls, various surgeons with no evaluation of technique, time of year.
Owings	10%	1%	No controls, no criteria for bleeding.
Peele	3% Av. Op. Time, 19.99 Min.	2.2% 17.39 Min.	No statistical significance.
Orzac	1.9%	0%	Used penicillin with Adrenosem, no evaluation of effect of penicillin on bleeding, no statistical significance. What were criteria that permitted no bleeding in 500 cases

There is no effect upon coagulation or bleeding time. There is no contraindication of any sort. In spite of the fact that its chief activity is upon capillary permeability which is ordinarily not a problem in surgery, there has been considerable literature[3-9] to indicate its effectiveness in a variety of surgical procedures.

Table I is a compilation of the recent literature. The authors admit to weakness in their statistical data, but all have the impression that adrenochrome helped reduce bleeding. The present study developed as a controlled evaluation that would have statistical significance.

METHODS.

The following conditions were carefully observed to limit the variables which could control blood loss:

1. Two-hundred consecutive cases limited to children up to age 12 were used. Eleven were discarded from the data due to errors in measurement at time of surgery.

2. Standard premedication, consisting of codeine and atropine, according to age and weight.

3. Ether anesthesia by insufflation.

4. Random selection of patient without the knowledge of the surgeon. This was done by the ward nurse who was instructed to give the Adrenosem® with the premedication to alternate cases.

5. Elimination of cases with factors which may influence bleeding. These included recent infection requiring antibiotics, rheumatic fever and kidney disease.

6. Consistent surgical technique, using the Crowe-Davis position and McIvor gag. The adenoids were removed with curette and punch forceps. One sponge saturated with 10 per cent tannic acid jelly was used to pack the nasopharynx in all cases. Tonsillectomy was done by dissection and snare. Upper pole bleeders were routinely clamped and tied before dissecting. Those remaining were clamped when necessary. An average of four to six ties were routinely used. No record of the number of ties was kept. Adrenosem® has no reported effect on vessels larger than capillaries.

7. Careful measurement of blood loss. This was done by using only a measured amount of citrated water for irrigation of the tubing during and upon completion of surgery.

8. Definition of bleeding. There is no consistency in the literature regarding the criteria of "unusual bleeding." The following were used:

a. Persistent puddling of blood at completion of surgery.

b. Need for agents other than the ties and one Amertan sponge to control bleeding. These agents included 1 per

cent Ferric-chloride, Monsel's solution, 1 per cent Neosynephrine or Epinephrine, postnasal pack or transfusions.

c. Postoperative oozing during the first 24-hour period requiring examination, even if not treated.

These criteria were deliberately made rigid to record the cases of oozing which might otherwise be missed. The cases of oozing, if assumed to be due to capillary leakage, would be those most likely to be helped by the Adrenosem®.

TABLE II.

Average Blood Loss in Each Age Group.

Age in Yrs.	No. of Cases		Vol. of cc. Blood Loss	
	Control	Adrenosem	Control	Adrenosem
1 to 2	5	4	30	38.8
2 to 3	7	13	42.9	41.9
3 to 4	12	15	41.3	64
4 to 5	19	19	89.5	56.3
5 to 6	15	15	93.0	75.7
6 to 7	15	15	77.7	99.2
7 to 8	5	7	87	103.6
8 to 10	9	6	111	95
10 to 12	4	6	93.8	130
Total Cases	91	98		

RESULTS.

Tables II and III graphically represent the results of the study.

1. There were insufficient cases within each age group for significant statistical evaluation. The lack of consistent difference within the various age groups between the control and treated cases indicates no correlation between age and response to Adrenosem®.

2. In the 98 control cases there were seven with excessive bleeding, making a total of 7.7 per cent as compared to 8.1 per cent in the 98 medicated ones. This likewise is of no significance, and agrees with the clinical impression gained at surgery. It was impossible to differentiate control and Adrenosem® cases by the ease of the operation.

3. Statistical interpretation is shown in Table IV. There

TABLE III.

Complications and the Means of Their Control in Each Age Group.

Age in Yrs.	Complications	
	Control	Adrenosem
1 to 2	0	0
2 to 3	0	0
3 to 4	0	1. Post nasal pack. 2. Ooze (1% Neosyn.).
4 to 5	Aden. ooze	0
5 to 6	1. Aden. ooze ($FeCl_3$) 2. General ooze	0
6 to 7	Post-op. bleeding	1. Aden. ooze during surgery and post op. 2. Aden. ooze. 3. Many ties, aden. ooze.
7 to 8	Aden. ooze	General ooze.
8 to 10	1. Post nasal pack and transfusion 2. Ooze.	
10 to 12		1. Gen'l ooze (Adrenoline, Monsels) 2. Prof. bleeding (Neosyn., $FeCl_3$)
Complications Total Cases	7	8
Per Cent	7.7%	8.1%

TABLE IV.

Statistical Evaluation of Experimental Results.
Adenosem Study of T. & A.
189 Children, Ages 11 mos. to 12 yrs.—May, 1956 to Aug. 1957.

	Total	Sex	Mean Volume	S.D.*	S_n†	S_n of Diff. of Means‡
Control	91	M 54 F 40	78.2 cc.	45.3 cc.	4.8 cc.	
						6.5 cc.
Adrenosem	98	M 55 F 43	76.1 cc.	42.9 cc.	4.4 cc.	

Conclusion—No Statistical Significance.

*S.D.—Standard deviation of mean; expected variability (±) of individual samples.

†S_n—Standard error of the mean; expected variability (±) of the means.

‡S_n Standard Error of Difference of Means—The difference to be expected by chance.

was a difference of 2.1 cc. in blood loss between the two groups. This is well within the limits anticipated by pure chance (*i.e.*, standard error of the difference of the means—6.5 cc.) and is, therefore, not significant. There was likewise no correlation between sex and bleeding; however, when the bleeding of all cases is compared to age (see Fig. 1), a

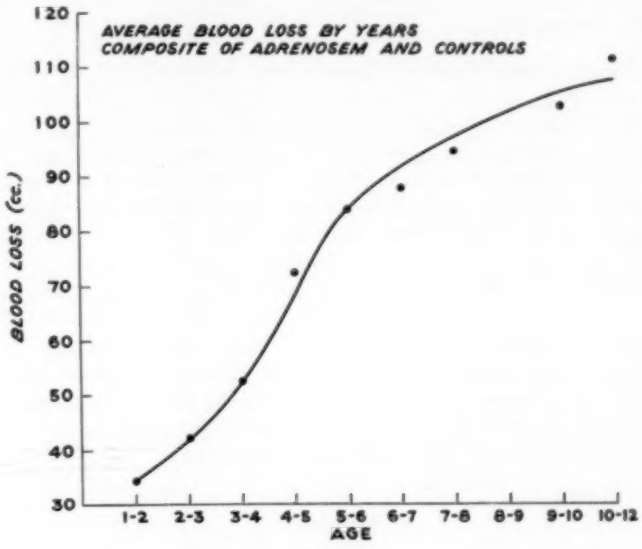

Fig. 1.

sigmoid curve is derived. This is graphic evidence of the common observation that bleeding increases with age until a plateau is reached.

4. As a matter of interest, the bleeding and coagulation times were compared to the blood loss by the X^2 method. This showed no correlation between the tests and the amount of blood loss.

DISCUSSION.

It is apparent that there is no discernible difference, clini-

cal or statistical, between the control and treated cases of this series. In all fairness it must be emphasized that this study evaluates the Adrenosem® action in a series of cases where bleeding was kept to a minimum. The bleeding was adequately controlled by careful surgical technique, including unimpaired visualization, clear airway, minimal trauma, avoidance of extra-capsular dissection, immediate tying of blood vessels and meticulous removal of lymphoid tissue from the nasopharynx.

Proctor and Douglass[10] have shown a 1 per cent incidence of bleeding in over 1600 cases without special medication. This minimal figure as compared to the present study is probably attributable to a difference in criteria of bleeding. In any case, the significance is not in the figure itself but in the fact that highly creditable results were obtained by surgical management alone. They concluded that "as yet there is no adequate justification for the widely accepted belief that the so-called hemostatic drugs . . . . will reduce the incidence of postoperative bleeding below that figure to be expected with careful surgical technique alone." This study corroborates this opinion and emphasizes again the need for critical evaluation of therapeutic agents. The value of the hemostatic drugs is so difficult to assess that only the most careful analytical studies should be considered adequate.

SUMMARY.

1. The anti-hemorrhagic effect of adrenochrome semi-carbazone (Adrenosem®) was studied on a series of 189 adenotonsillectomies of which 91 were unmedicated controls.

2. No clinical or statistical difference could be discerned between the treated and control cases.

3. Evidence was presented that surgical technique is statistically of greater importance in decreasing blood loss than any known medication.

4. An incidental finding was the lack of correlation between the amount of blood loss and the bleeding and coagulation time.

BIBLIOGRAPHY.

1. Fulton, G. P.; Luty, B. R.; Shulman, M. H., and Arendt, K. A.: "Moccasin Venom as a Test for Susceptibility to Petechial Formation." Read at the 121st Annual Meeting of the A.A.A. Science, Berkeley, Calif., Dec. 26-31, 1954.

2. Arendt, K. A.: "Moccasin Venom as a Test for Petechial Susceptibility in the Cheek Pouch and Mesoappendix of the Hamster." Ph. D. Dissertation, Boston University Graduate School, 1955.

3. Sherber, D. A.: The Control of Bleeding. *Amer. Jour. Surg.*, 86:331-335, 1953.

4. Owings, C. B.: The Control of Postoperative Adenoid Bleeding with Adrenosem. The Laryngoscope, 65:21-24, 1955.

5. Peele, J. C.: Adrenosem in the Control of Hemorrhage from the Nose and Throat. *Arch. Otolaryngol.*, 61:450-464, 1955.

6. Riddle, A. C.: Adrenosem Salicylate, a Systemic Hemostatic. *Oral Surg., Med. and Path.*, 8:617-620, 1955.

7. Ryan, E.: Medical Care of the Child Patient Before and After Adenoidectomy and Tonsillectomy. *N. Y. St. Jour. Med.*, 56:886-887, 1956.

8. Bacala, J. C.: The Use of the Systemic Hemostat Carbazochrome Salicylate. *West. Jour. Surg., Obstet. and Gynec.*, 64:88-95, 1956.

9. Coyle, J. E.: Analysis of Blood and Vascular Factors in the Prophylaxis of Tonsillo-Adenoidal Hemorrhage. The Laryngoscope, 67:1029-1061, 1957.

10. Proctor, D. F., and Douglass, C. C.: Bleeding Following Tonsil and Adenoid Operations. *Trans. Amer. Acad. Ophthal. and Otol.*, 592-597, July-Aug., 1958.

THREE NEW ADENOID PUNCH FORCEPS FOR ADENOIDECTOMY.

MERRILL LINEBACK, M.D.,

College Park, Ga.

Adenoidectomy with or without tonsillectomy is the most frequently performed surgical operation, being largely a hit and mostly a miss affair. In no other field of surgery, obstetrics excepted, is the field of vision so limited and, with respect to the adenoids, are the results as poor. Not a few surgeons pride themselves on the rapidity of technique and the number of extirpations per hour; seldom do they concern themselves with the long term effects of a poorly performed adenoidectomy. These are "regrowth" and continued Eustachian tube blockage maintaining deafness in a child, either as a result of a "collapsed" ear drum or fluid in the middle ear, or both.

The adenoids and tonsils do not "regrow" if they are completely removed the first time. It is the tags that remain which hypertrophy after repeated reinfections and present later as "regrowths." Cysts in the adenoid region of the adult are often unsuspected until the adenotome is used over a smooth appearing and "flat" nasopharynx. The author has had two cases of adult cysts which had inspissated putty-like material in the uncovered adenoid; one was a patient 22 years old, and the other was 24 years, both females.

Surgeons who work with congenital deformities of the heart have found that a dry field and direct vision are much to be preferred over the blind techniques.

Direct vision during adenoidectomy is never precise. It is impossible to throw light into all reaches of the crypts and fossae of the nasopharynx, particularly in the region of the choanae; but with the large No. 6 laryngeal mirror, which has been previously warmed, the entire region of the naso-

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Feb. 15, 1959.

pharynx can be inspected serially and systematically with full illumination reflected from the mirror. Even with the soft palate retracted it is impossible to examine the choanae adequately without use of the mirror. The author routinely uses the one-inch wide Penrose tubing which has been secured through the nose by a previously introduced 14 Fr. soft rubber catheter. Light tension is then placed both on the remaining portion of the Penrose through the mouth and the end coming through the nose, and both ends are secured with a short Halstead forceps to prevent slippage. The one-inch width allows a more even pressure application to the soft palate and effectively elevates it without tearing, which often happens when the catheter alone is used. The device frees both hands for use of the warmed mirror and the punch forceps.

The surgeon is seated at the head of the patient who is in the supine Rose position with the head low and practically in the lap of the operator. The Davis or McIvor mouth gag is used during the tonsillectomy and remains in place during the adenoidectomy. The opposite end of the tongue blade is suspended from the edge of the Mayo tray which is over the chest of the patient. This frees the assistant's or anesthetist's hand for more pressing duties. Since the usual tongue blade used frequently obscures the lingual tonsil and the base of the tonsil fossa itself, the operation is finished with the Jenning's gag and separate tongue depressor (thin) so that these areas may be inspected more thoroughly, and the surgeon can thus rest assured that the tonsillectomy and adenoidectomy is complete. With complete removal of the adenoids there is less chance of postoperative hemorrhage. If this eventuality is anticipated, the author used either the Fox nasal balloon or the 30 cc. Bardek bag into which 10 to 15 cc. of air has been put via syringe and No. 26 needle. Removal of the system is simply by letting the air out with a No. 20 needle and pulling the catheter and collapsed bag out through the nose.

The patient, of whatever age, will tolerate the air balloon much better than the usual gauze adenoid packing which soon becomes very odoriferous and most difficult to remove through the mouth.

DESCRIPTION OF FORCEPS.

The forceps labeled 1. in Fig. 1 is a reverse curve, and has a reverse bite for use in the choanae. The punch itself is triangular in shape, and the three sides are sharp; each basket of the punch has two parallel wires as a catch for the bits of tissue as they are punched out.

No. 2 forceps is curved toward the operator when held in the right hand. The punch head is so opened that it bites to

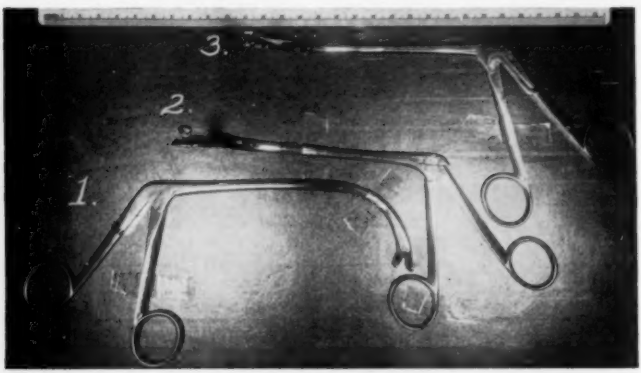

Fig. 1. Adenoid punch forceps. 1. is reverse curve-reverse biting; 2. is full 90 degree curved toward the right-handed operator, biting downwards for the left Rosenmueller fossa; 3. is full 90 degree curved toward the operator, biting upward for the right fossa. All three are 15 cms. in length from the fulcrum to the head.

the left when placed in the nasopharynx and is used chiefly in the left Rosenmueller fossa. The same instrument can be made for a left handed operator by curving the head in the opposite or mirror image direction. In that case the instrument would be used in the right fossa.

No. 3 forceps is again curved toward a right handed operator but with the head biting upwards, and is thus chiefly used in the right Rosenmuelled fossa. If the instrument is designed for a left handed operator, it would be used in the left fossa. All three forceps are 15 cms. long from the fulcrum to the head itself.

SUMMARY.

Three new modifications of adenoid punch forceps are presented with indications for their use. The author's routine for complete tonsillectomy and adenoidectomy is briefly described. With complete removal of tissue there is less possibility for postoperative hemorrhage, and "regrowth" chances are thus eradicated. These forceps may also be used as biopsy forceps when a tumor of the fossae or choanae is suspected. They are manufactured by the Storz Instrument Co., 4570 Audubon Ave., St. Louis 10, Mo.

124 West Princeton Ave.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1960.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

President: Dr. J. J. Longacre, 1503 Carew Tower, Cincinnati, O.
President-Elect: Dr. D. C. Samuel Pruzansky, D.D.S., 840 So. Wood St., Chicago, Ill.
Secretary-Treasurer: Dr. Spriestersbach, Ph.D., Deptartment of Otolaryngology, University Hospital, Iowa City, Ia.
Meeting: Palace Hotel, Denver, Colo., May 12-14, 1960.

AMERICAN BOARD OF OTOLARYNGOLOGY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee Dr., Syracuse 10, N. Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Ia.
Meeting: Chicago, Ill., October, 1960.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Verling K. Hart, 107 W. 7th St., Charlotte, N. C.
Vice-President: Dr. Daniel C. Baker, Jr., 903 Park Ave., New York, N. Y
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3, Pa.
Treasurer: Dr. Charles M. Norris, 3401 Broad St., Philadelphia, Pa.
Meeting: Deauville Hotel, Miami Beach, Fla., March 15-16, 1960 (Afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. W. J. McNally, Montreal, Canada.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.
Treasurer: Dr. Francis E. LeJeune, New Orleans, La.
Editor, Historian, and Librarian: Dr. Edwin N. Broyles, Baltimore, Md.
Meeting: Deauville Hotel, Miami Beach, Fla., March 18-19, 1960.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
President-Elect: Dr. Fletcher D. Woodward, 400 Locust Ave., Charlottesville, Va.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Treasurer: Dr. K. M. Day, 121 University Pl., Pittsburgh, Pa.
Annual Meeting: Deauville Hotel, Miami Beach, Fla., March 13-19, 1960.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Paul H. Holinger, Chicago, Ill.
Vice-Chairman: Dr. Lawrence R. Boies, Minneapolis, Minn.
Secretary: Dr. Walter E. Heck, San Francisco, Calif.
Representative to Scientific Exhibit: Dr. Walter H. Maloney, Cleveland, Ohio.
Section Delegate: Dr. Gordon F. Harkness, Davenport, Ia.
Alternate Delegate: Dr. Dean M. Lierle, Iowa City, Ia.
Meeting: Miami Beach, Fla., June 13-17, 1960.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. R. C. Martin, 384 Post St., San Francisco 8, Calif.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14, Minn.
Meeting: Deauville Hotel, Miami Beach, Fla., March 13-14, 1960.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worchester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Kenneth H. Hinderer, 402 Medical Arts Bldg., Pittsburgh 13, Pa.
Secretary: Dr. Robert M. Hansen, 1735 No. Wheeler Ave., Portland 17, Ore.
Annual Clinical Session: October 8-9, 1959, Illinois Masonic Hospital, Chicago, Ill.
Annual Meeting: October 10, 1959, Belmont Hotel, Chicago, Ill.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Oscar J. Becker, Chicago, Ill.
Vice-President: Dr. Sam H. Sanders, Memphis, Tenn.
Treasurer: Dr. Joseph C. Miceli, Brooklyn, N. Y.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28, N. Y.
Meeting: Chicago, Ill., October, 1960.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Michael H. Barone, Buffalo, N. Y.
President-Elect: Dr. Walter E. Owen, Peoria, Ill.
Vice-President: Dr. Frank P. Powers, Raleigh, N. Car.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
Annual Meeting:

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Alberto Gallo.
First Secretary: Dr. Alfredo Martinelli.
Second Secretary: Dr. Guedes de Melo Neto.
Librarian-Treasurer: Dr. L. de Souza Queiroz.
Editors for the Archives of the Society: Dr. Antonio de Almeida, Dr. Gabriel Pôrto, and Dr. Roberto Franco do Amaral.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Md.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Rd., Halifax, Nova Scotia.
Meeting: Sheraton-Brock Hotel, Niagara Falls, Ontario.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. William F. Hubble, Decatur, Ill.
President-Elect: Dr. Charles D. Sneller, Peoria, Ill.
Vice-President: Dr. Edgar T. Blair, Springfield, Ill.
Delegate at Large: Dr. G. Leroy Porter, Urbana, Ill.
Secretary-Treasurer: Dr. Clarence A. Fleischli, Springfield, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. George Woodruff, Woodruff Clinic, Joliet, Ill.
Vice-President: Dr. Linden Wallner, 122 So. Michigan, Chicago, Ill.
Secretary-Treasurer: Dr. Robert Lewy, 25 East Washington St., Chicago 2, Ill.
Meeting: First Monday of each month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

COLORADO OTOLARYNGOLOGY SOCIETY.

President: Dr. James T. Blair, Denver, Colo.
Vice-President: Dr. James Rigg, Grand Junction, Colo.
Secretary: Dr. Will P. Pirkey, Denver, Colo.

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. John E. Arthur.
Secretary: Dr. M. L. Battles.
Meetings: First Monday of October through May, University Club, Columbus, O.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edward A. Newell.
Vice-President: Dr. Thomas M. McCrory.
Secretary-Treasurer: Dr. James L. Baldwin, 1627 Medical Arts Bldg., Dallas, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of the Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. Dekle Taylor, Jacksonville, Fla.
President-Elect: Dr. Kenneth S. Whitmer, Miami, Fla.
First Vice-President: Dr. William H. Anderson, Jr., Ocala, Fla.
Second Vice-President: Dr. Marion W. Hester, Lakeland, Fla.
Secretary-Treasurer: Dr. Joseph W. Taylor, Jr., 1 Davis Blvd., Tampa 6, Fla.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Van D. Rathgeber.
Vice-President: Dr. William Skokan.
Secretary-Treasurer: Dr. Paul Rockwell.

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Mariano C. Caballero.
Vice-President: Dr. Joseph Freeman.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting: Quarterly in March, May, October and December on the second Thursday of the month, 6:30 P.M., at the McAllister Hotel, Miami, Fla.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Fred D. Hollowell, Lamar Life Bldg., Jackson, Miss.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting:

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairmen in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Monterey 47, Mexico 7, D. F.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.

(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Ohrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. David D. DeWeese, 1216 S. W. Yamhill St., Portland 5, Ore.
Secretary-Treasurer: Dr. Paul B. Myers, 223 Medical Dental Bldg., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Henry Thiele Restaurant, 23rd and W. Burnside, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

President: Dr. E. P. Fowler, Jr., 180 Fort Washington Ave., New York 32, New York.
Secretary-Treasurer: Dr. Arthur L. Juers, 1018 Brown Building, Louisville 2, Ky.
Meeting: Palmer House, Chicago, Ill., October, 1960.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. John F. Tolan, 1118 - 9th Ave., Seattle 5, Wash.
Secretary-Treasurer: Dr. Homer E. Smith, 686 Twelfth Ave., Salt Lake City, Utah.
Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President Dr. John J. O'Keefe.
Vice-President: Dr. Joseph P. Atkins.
Secretary: Dr. William A. Lell.
Executive Committee: Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, Dr. William A. Lell, Dr. William J. Hitschler, and Dr. Chevalier L. Jackson.

PHILIPPINE SOCIETY OF OTOLARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Macario G. Tan, 426 Evangelista, Manila, P. I.
Vice-President: Dr. Ariston G. Bautista, 460 Isaac Peral, Manila, P. I.
Secretary-Treasurer: Dr. Angel Enriquez, American Hospital, Aduana St., Manila, P. I.

PITTSBURGH OTOLOGICAL SOCIETY.

President: Dr. Emory A. Rittenhouse, 203 Masonic Bldg., McKeesport, Pa.
Vice-President: Dr. Carson S. Demling, 513 Jenkins Bldg., Pittsburgh 22, Pa.
Secretary-Treasurer: Dr. Clyde B. Lamp, 8101 Jenkins Arcade, Pittsburgh 22, Pa.

PORTUGUESE OTORHINOLARYNGOLOGICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

Meeting: University of Vienna, August 29 to September 1, 1960.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

President: Dr. F. T. Land, 13 Newton Place, Glasgow, C. 3.
Secretary-Treasurer: Dr. J. F. Birrell, 14 Moray Place, Edinburgh.
Assistant Secretary: Dr. H. D. Brown Kelly, 11 Sandyford Place, Glasgow, C. 3.

SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA.

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marafiés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Monterrey 47-201
Mexico 7, D. F.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela.
Treasurer: Dr. Benito Madariaga.
First Vocal: Dr. Rafael González.
Second Vocal: Dr. Juan Oberhauser.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villers.
Vice-Presidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID.

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael Garcia Tapia.

SOCIEDAD VENEZOLANA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Gabriel Briceño Romero.
Vice-Presidente: Dr. Silvestre Rincón Fuenmayor.
Secretario General: Dr. Oscar Bustamante Miranda.
Tesorero: Dr. Arturo Marrero Gómez.
Vocales: Dr. Miguel Octavio Russa, Dr. Benjamin Briceño, Dr. Oscar Gonzalez Castillo.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DO RIO GRANDE DO SUL.

President: Dr. Ivo Adolpho Kuhl.
Secretary: Dr. Decio Lisboa Castro.
Treasurer: Dr. Jorge Valentin.

SOCIEDAD PANAMENA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Manuel Preciado.
First Vice-Presidente: Dr. Alonso Roy.
Second Vice-Presidente: Dr. Carlos Arango Carbone.
Secretario: Dr. María Esther Villalaz.
Tesorero: Dr. Ramòn Crespo.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Lt. Col. Stanley H. Bear, USAF (MC), USAF Hospital, Maxwell (Air University), Maxwell Air Force Base, Ala.
Secretary-Treasurer: Capt. Maurice Schiff, MC, USN, U. S. Naval Hospital, Oakland, Calif.
Meeting:

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. F. R. Price, 118 Rutledge Ave., Charleston, S. C.
President-Elect: Dr. L. D. Lide, 161 W. Cheves St., Florence, S. C.
Vice-President: Dr. R. E. Livingstone, 1505 Main St., Newberry, S. C.
Secretary-Treasurer: Dr. Roderick Macdonald, 330 E. Main St., Rock Hill, S. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. V. Eugene Holcombe, Charleston, W. Va.
Chairman-Elect: Dr. G. Slaughter Fitz-Hugh, Charlottesville, Va.
Vice-Chairman: Dr. George M. Haik, New Orleans, La.
Secretary: Dr. Mercer G. Lynch, New Orleans, La.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin Sheppard, 301 Medical Arts Building, Richmond, Va.
President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Va.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Nime K. Joseph, Wheeling, W. Va.
President-Elect: Dr. John A. B. Holt, Charleston, W. Va.
Vice-President: Dr. William K. Marple, Huntington, W. Va.
Secretary-Treasurer: Dr. Albert C. Esposito, Huntington, W. Va.
Director for Two Years: Dr. James T. Spencer, Charleston, W. Va.

NOTICE TO CONTRIBUTORS

THE LARYNGOSCOPE reserves the right of exclusive publication of all articles submitted. This does not preclude their publication in Transactions of various Societies.

Manuscripts should be typewritten, double spaced, on one side of paper only and with sufficient margins to allow for corrections.

Author's name and city should appear directly under title on first page; street address at end of article.

All prints or photographs to be submitted in black and white, in good sharp contrast. Good halftones depend upon clear photographs. Line drawings for zincs to be in black and white. Colored inks or red or blue quadrille rulings will not reproduce.

References should be complete: author's surname, initials, title of article, Journal, volume, page, month, year.

Six illustrations will be furnished for each article without cost to author. Authors will please limit illustrations to six or assume the expense of additional illustrations.

Proofs will be submitted to authors for corrections. If these are not returned, articles will be published as corrected in this office.

Reprints will be furnished at the following prices:

WITHOUT COVER

	250 Copies	500 Copies	1000 Copies	2000 Copies
Four Pages	$ 19.25	$ 23.00	$ 30.75	$ 44.50
Eight Pages	33.50	42.75	58.50	83.00
Twelve Pages	47.00	60.75	86.25	131.50
Sixteen Pages	61.00	78.75	98.75	146.75
Twenty Pages	76.00	96.25	129.50	187.25
Twenty-four Pages	88.75	112.50	150.00	217.25
Twenty-eight Pages	97.50	123.25	162.25	233.50
Thirty-two Pages	115.00	139.75	180.00	267.00

WITH COVER

Four Pages	$ 37.25	$ 46.50	$ 61.50	$ 88.75
Eight Pages	51.50	66.25	89.25	127.25
Twelve Pages	65.00	84.25	117.00	175.75
Sixteen Pages	79.00	102.25	129.50	191.00
Twenty Pages	94.00	119.75	160.25	231.50
Twenty-four Pages	106.75	136.00	180.75	261.50
Twenty-eight Pages	115.50	146.75	193.00	277.75
Thirty-two Pages	133.00	163.75	210.75	311.25

Express charges will be paid by consignee.